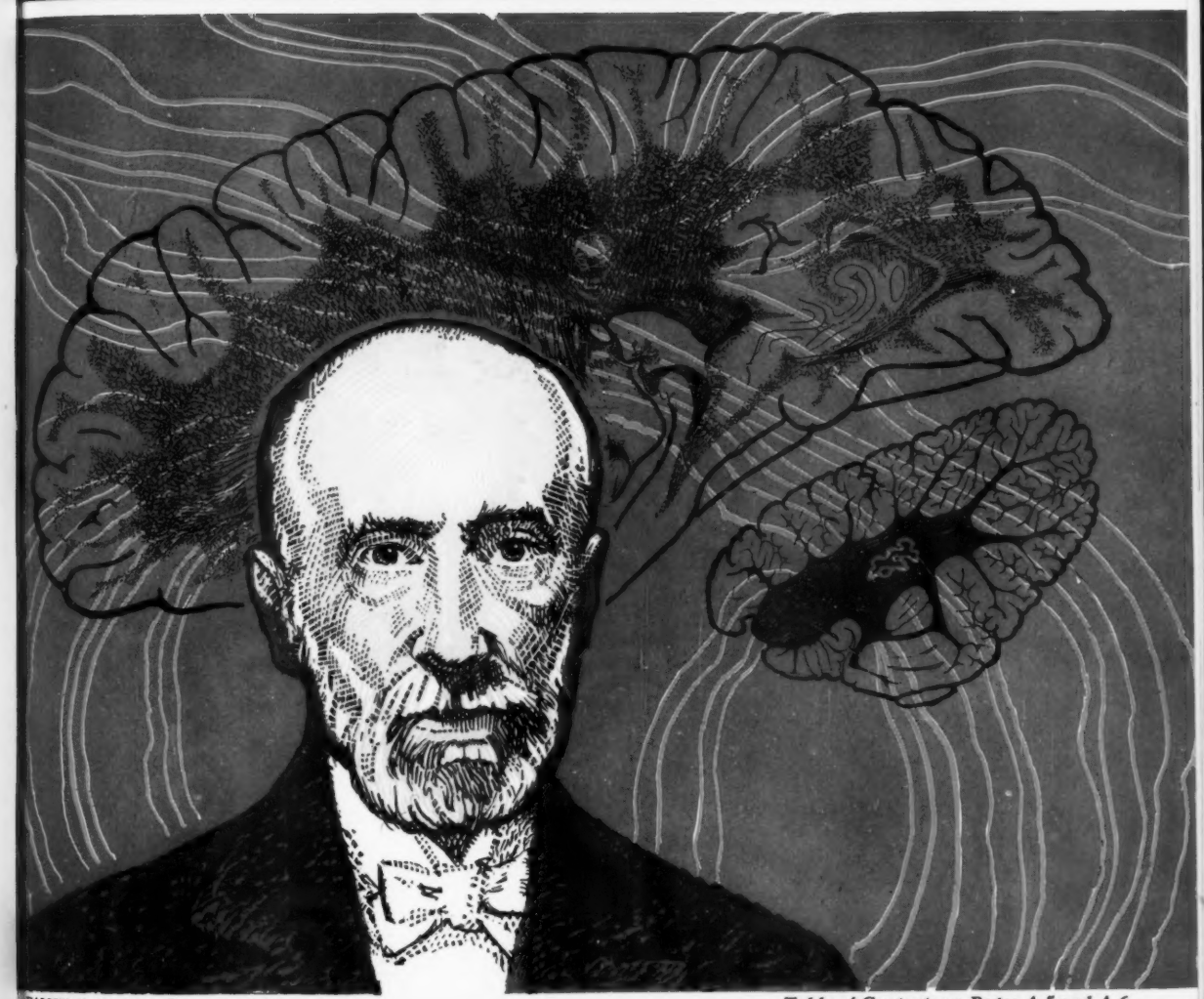
POSTGRADUATE MEDICINE



RAMON Y CAJAL—*See page 264*

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The Location of the Mental Functions of the Brain

LELAND B. ALFORD*

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Evidence both from the literature and from my own cases indicates that dementia (or confusion) is produced from the region of the thalamus and from no other part of the brain. A number of variable complicating factors are found to account for the various clinical pictures which are usually assigned to the cortex and which are therefore spurious.

I ALWAYS speak of cerebral localization with some trepidation because the ideas expressed are so at variance with accepted tenets that aspersions are cast upon the cherished traditions concerning the cerebral cortex.

L. B. ALFORD

Under the circumstances perhaps it is well to mention a few historical sequences. My first paper on cerebral localization was read in 1932, and the thesis was that clouding of consciousness or dementia could be localized.[1]

To understand how this thesis came about, it is necessary to refer to some of the difficulties, academic and practical, confronting the inexperienced investigator of cerebral localization. There is first the contradiction in findings, when the occasional negative or normal picture crops up to confuse a syndrome defined and localized over the years. Then there are the elusive "centers," which like a will-o'-the-wisp somehow just escape capture when efforts are made definitely to pin them down. And still worse is the rather ridiculous circumstance that haphazard investigation has piled center upon center, as it were, in a single part of the brain—as in the case of the supramarginal-angular region for which Critchley was able to collect some 23 syndromes.[1a]

In addition are the practical difficulties confronting the budding investigator. The various clinical pictures he wishes to localize in the patient before him—unevenness in mental performance, aphasia, memory failure—soon prove to be so vague, so variable from day to day (even from hour to hour) and so temporary as to defy any exact relating of data. Certainly to localize, at least early in one's experience, there must be something definite to work with.

Thus in my own case it happened that confusion or dementia was the clinical picture chosen for localization. Among all psychiatric symptoms, this one is the most definite. Sooner or later it is recognized with some certainty by the experienced specialist in a great majority of instances. Unfortunately no one term traces that purely psychologic picture of mental dimming through all the infections, intoxications, exhaustions and organic conditions in which it occurs. Hence the various terms—clouding of consciousness, confusion, disorientation, dementia—which it is necessary to employ in trying to describe the very definite and exact defect of

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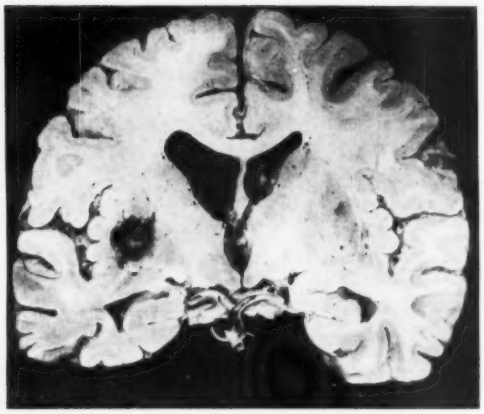

FIGURE 1. Vascular softening, 1 cm. in diameter, located in the left lenticular nucleus. The symptoms may be roughly divided into four parts, each of two weeks' duration: (1) a period of deep confusion; (2) a period of improvement; (3) a relatively stable period of orientation but of dullness or stupidity, and (4) the time of physical decline ending in death. This sequence is supposedly related to the effect of complicating reactions about the lesion affecting a nearby center, presumably in the thalamus, the reactions slowly subsiding until the final decline starts.[9]

awareness or alertness that is meant. I often use several of these terms interchangeably in order to approximate the meaning intended.

The result of this manner of approach was that my first paper, 20 years ago, supported the thesis that confusion or dementia could be localized. It was pointed out that patients with aphasia or other symptoms of organic lesions in cortical areas still were capable of complex mental feats when given a fair chance and investigated in an offhand, sociable manner rather than being subjected to a police-like third degree, which only confused and distressed. On the other hand, it was stated that since one knew from experience that focal lesions sometimes cause early and deep dementia, it could only follow that confusion could be localized. This train of thought seemed logical enough but, perhaps because cases with postmortem correlations were lacking, that first paper was received with something less than acclaim.

The next paper, published in 1937, followed five years devoted to collecting parallel clinical and anatomic data, with positive and control bearings.[2] The task was really more difficult

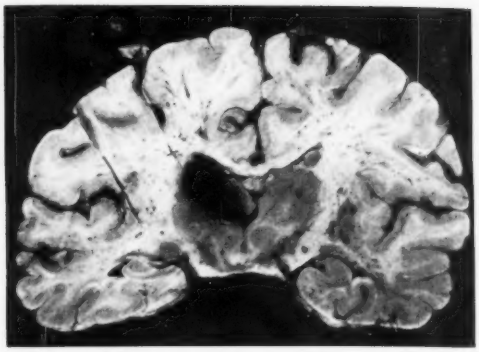

FIGURE 2. A lesion consisting of a hemorrhage almost filling the left thalamus. The patient survived for a month, remaining in good condition for three weeks of the time. The confusion was complete.[2]

than it might seem at first glance: The clinical picture had to be found, the patient had to be in fair general condition, he had to survive long enough for initial shock effects to pass and then die while still under treatment. Moreover, an autopsy permit had to be obtained, the designated pathologist had to refrain from cutting up or mauling the brain on the spot and, finally, the lesion had to turn out to be single and uncomplicated. Obviously the odds in favor of the hopeful investigator are not large. But in my own case, perhaps with some luck, the desired cases did come along as will be seen from the illustrations and descriptions. The final consequence of the data—both that personally collected and that drawn from the literature, which fortunately in the meantime also was increasing—is the conclusion as to the location of the mental processes.

CONFUSION OR DEMENTIA FROM THALAMIC LESIONS

EVIDENCE in the literature bearing on the localization of confusion which has been slow in accumulating because of lack of consistent investigation is now impressive. One very helpful paper and, incidentally, one of the finest papers in neurologic literature is that of the English workers, Smyth and Stern (1938).[3] These writers discovered a type of glioma that arises in the subependymal layer covering the third ventricular surface of the thalamus. In their

FIGURE 3. This drawing represents a slit of vascular softening running anteroposteriorly through about the middle of the left basal nuclei. The patient survived for two weeks, most of the time in good vigor. The confusion was profound. (From Alford, L. B.[2] Reproduced courtesy *American Journal of Psychiatry*.)

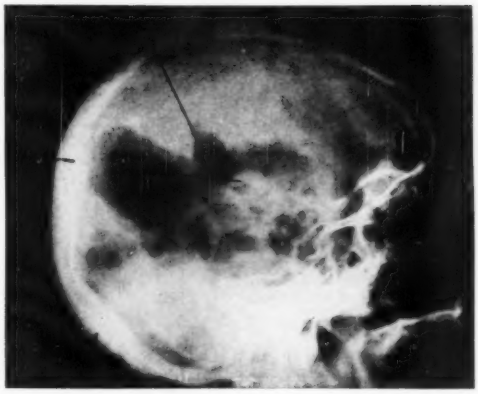

FIGURE 4. An enormous cavity (as revealed by air) occupying almost exactly the speech area of the cortex and immediate subcortical parts. The patient showed marked aphasia but continued to direct the detail of rather extensive banking and farming interests; his mentality was excellent.[2]

series of some 4 cases, it was conspicuous that dementia was one of the very first symptoms and became extreme, while with tumors arising in the capsule or with tumors in general, this symptom arises much later, if at all, before actual coma develops. Nielsen of Los Angeles, a tireless investigator, has reported a number of cases and emphatically supports the localization of dementia in the thalamus, as shown by discussions and abstracts of exhibitions and papers.[4] Walther of Germany reported finding a small cystic area in the thalamus of a patient with delirium.[5] Grunthal of Germany and Stern of England each reported cases of primary degenerations limited to the thalamus that are particularly helpful because they are free of the complicating "neighborhood reactions."[6,7] In addition, the late Dr. Walter Dandy of Johns Hopkins reached the conclusion that the same symptom arises from lesions in the corpus striatum, which of course is not far away from the thalamus.[8] It is particularly conclusive that so many observers, working independently, should discover a specific symptom-lesion association previously unsuspected.

The illustrations and references to the literature demonstrate the great contrast in the effect on the mental life in general and the production of mental confusion in particular, between thalamic injuries and cortical lesions, even when these lesions involve what are regarded as the most vital areas. There seems to be no factor that could account for this divergence except the location of the injury and hence the conclusion as to the localization of dementia.

The sizes and relative mildness of the lesions illustrated in Figures 1, 2 and 3 should be contrasted with the large areas of destruction pictured in Figures 4, 5 and 6. The mentality of the latter group was capable of high performance. Only the parts of the cortex that are supposedly vital in one way or another are shown, but all other parts of the cortex display the same results.

COMPLICATING FACTORS

The very obvious lesion-symptom relationships just depicted certainly would long since have been detected and evaluated, had there not been complicating factors. There are several of these deceptive aspects, attitudinal, psychologic and physical, all of which are important.

Speech disturbances—The assumption has been made that there is a distinct function of speech, whose disturbance leads to one or another of the numerous forms of aphasia that have been described. The apparent isolation and relative severity of some speech disturbances are indeed striking; but upon closer analysis, it is no exaggeration to state that no form is at all isolated from other psychic functions, is in any sense stable or demarcated or can be localized to any degree. These assertions are clearly sup-

FIGURE 5. A large glioma occupying the supramarginal-angular area of the left cerebral hemisphere. The patient, a chemistry professor, met his classes until two weeks before the operation, when a mild aphasia forced action. Subsequent to the operation, he not only resumed teaching for a time but also appeared "entirely normal" to his wife. This illustration is shown because, as mentioned in the text, many syndromes have been traced to this area, all of which evidently are spurious.

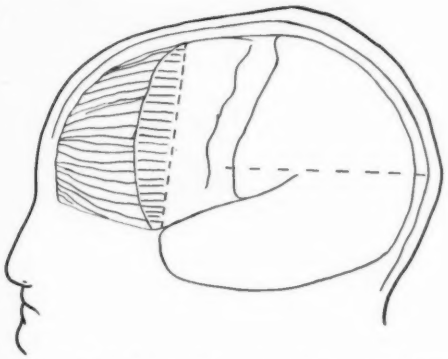

FIGURE 6. This figure shows the almost complete removal of both frontal lobes. The mental defects could be detected in neither psychologic tests of the patient nor in a follow-up of civilian and military performance covering some six years. This case, involving as it does the whole question of frontal lobe functioning, is discussed further in the text. (From Penfield, W.[10] and Hebb, D. O.[11] Reproduced courtesy of *Archives of Neurology and Psychiatry*.)

ported in Weisenburg's monograph.[12] Although it is not possible to go into details here, it should be said that upon analysis, all the aphasias resolve into combinations of circumstances, consisting of a suggestible and confusable mentality which sometimes is influenced by certain motor or sensorial involvements. Thus the whole construction of aphasia is accidental and spurious.

Neglect of the suggestive or hysteroid factor in association with cerebral lesions—For some reason it is usually assumed that all psychic features must be valid when there is cerebral injury present. In fact, the physical centers in the brain are affected through circulatory changes even when the lesion is distant, and mentally the patient lives in terror, with the very center of his existence—his mental integrity itself—directly threatened. Hence, the mentality in the organic case is open to all sorts of confusion and distortion.

For one example, the so-called nominal aphasia, in which names are the one element conspicuously forgotten, is almost obviously a compulsive forgetting of the one part of speech that is specific and immutable. First one name is forgotten during a tense moment, then fearful anticipation extends the forgetting to all names.

Further, Figure 5 illustrates an instance of a normal mentality associated with a lesion of the lower parietal area, in the face of the circumstance that some 23 syndromes have been localized in this area. The likely explanation is that the patients generally have a suggestible mentality and, as with any hysteric, easily elaborate fictitious symptoms to correspond with the type of examination administered. Much of the literature on these matters supports my belief.

Physical changes in the brain—This complication comprises a "hidden" or invisible reaction or type of edema that may spread widely in all directions from an injury or from surgical incisions, and may affect distant functions. Thus, at first, one cannot know whether the symptom is due to the visible lesion or to the hidden change affecting some distant part. This change can be verified from motor and visual comparisons.[13]

The most surprising application of this factor is in the case of the frontal lobe syndromes. In Figure 6 it was shown that, in Penfield and Hebb's case, removal of both frontal lobes was followed by no considerable mental defect. Whence, then, the confusions, the dementias, the unaccountable compulsions following frontal lobotomies and injuries that range downward in degree to the point of no defect? The explana-

tion is that of a physical, hidden and variable change in the tissues extending backward from the incision or injury to centers in the thalamus. This assumption accounts for the flat contradiction in findings as well as for their great variability.

Mental capacity—The idea that mental level corresponds to efficiency; instability, fatigability, confusability and variability are not essential parts of capacity, however much they may interfere with efficiency. One complex feat accomplished while the patient is fresh and relaxed gives the true level of mentality. Thus the patient who found the quotation of his stock on the financial page of the daily paper was judged to have essentially a well preserved mentality.

These four complicating factors clarify the fallacious importance that in the past has been attributed to the cerebral cortex. The symptoms associated with cortical lesions—the aphasias, the frontal lobe syndromes and the elaborate mechanisms of the parietal lobe—are spectacular and are an incentive to dissect the mental mechanism. But the true nature of these symptoms is revealed by the inability to localize them exactly, by their variability and, above all, by those cases which absolutely contradict them.

CONCLUSIONS

In the foregoing discussion it was brought out that clinicoanatomic cases indicate that relatively small and innocuous lesions in the thalamic region are capable of producing the psychologic defect present in conditions of confusion and dementia, while larger and even bilateral areas of destruction elsewhere in the brain are consistent with complex mental performance. After considering all conceivable complicating factors, it appears that only the locations of the lesions can be the source of this contrast in effect and, therefore, that the psychologic defect mentioned can be localized.

On the other hand, the elaborate constructions that have been assigned to various areas of the cortex, such as the aphasias and the parietal syndromes, were thought to be capable of interpretation not as valid symptoms but as combinations of circumstances, chief of which was an unstable, suggestible and confusable but not deficient mentality. The frontal lobe complex of symptoms could be rationalized with the negative finding depicted in Figure 6 only by assuming that the symptoms were caused by a tissue change extending backward from the injury to affect the centers in the thalamus. The cortex was robbed of any major participation in the production of essential mental defect.

Four complicating factors of various logical orders were described as the chief sources of erroneous reasoning in the past. Thus it appears that the whole matter of cerebral localization is susceptible to a new method of approach and to re-evaluation on the basis of the production of dementia or confusion by injury to certain parts of the thalamus.

Since dementia or confusion, whatever the degree or the cause, means a practical cessation of all mental activity above a very primitive level and, since injuries elsewhere than in the thalamus do not interfere with the performance of complex mental feats, it follows that mental activity is centered in the thalamus and, as the cases cited show, chiefly in the left thalamus.[14]

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Bleeding Diverticula of the Thoracic Esophagus

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Bleeding from diverticula of the thoracic esophagus can occur. An instance of pulsion diverticulum and an instance of traction diverticulum of the esophagus, which were apparently the cause of anemia, are reported. Both cases were treated by surgical excision of the diverticula.

DIVERTICULA of the thoracic esophagus are an infrequent cause of hemorrhage from the gastrointestinal tract. Esophageal diverticula are divided into pulsion, traction and pulsion-traction types. Traction diverticula are ordinarily related to an inflammatory reaction in a lymph node which becomes adherent to the esophagus. These sacculations are therefore most commonly found near the bifurcation of the trachea or in the vicinity of the hilum of the lung. Pulsion diverticula are less frequent and are found in the epiphrenic portion of the esophagus which includes the lower third. Diverticula in the midportion of the esophagus rarely give rise to symptoms and are usually discovered incidentally at postmortem examination.

James[1] states: "Diverticula anywhere in the thoracic esophagus are rare. The symptoms are chiefly dysphagia, retrosternal pain or discomfort and anorexia. There is usually an associated weight loss. The epiphrenic diverticulum occurs chiefly on the posterior wall of the esophagus and extends toward the right thoracic cavity." Barrett,[2] in a report of a case in which the diverticulum was successfully resected, cites 115 cases, including his own, from a study of medical literature.

Traction diverticula (of the midportion of the esophagus) rarely require surgical treatment because of the relatively few symptoms which arise during the patient's lifetime. Some diverticula in this region have been considered congenital. The infrequency of pulsion diverticula of the thoracic esophagus is pointed out by Harrington.[3] He states that he has operated on 216 patients with pharyngoesophageal diverticula in contrast to only 8 patients in a similar period of time with thoracic pulsion diverticula. According to Harrington the symptoms are chiefly those associated with the swallowing of food and may resemble those produced by the esophageal hiatal type of diaphragmatic hernia or those produced by a diverticulum of the cardiac end of the stomach. In the 8 cases which he repaired surgically and which are reported in his article, stress was not placed on previous hemorrhage or severe anemia related to the condition.

Symptoms from thoracic diverticula ordinarily do not arise until the patient is in middle life or beyond. Ulceration may occur following the retention of debris, of food and of foreign

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material in the esophagocele, a term which Harrington ascribes to Reeves in 1855. However, massive hemorrhage from ulceration of an esophageal diverticulum apparently is rare.

Here we report 2 cases of thoracic diverticula, one of the pulsion and one of the traction type, in which severe anemia and hemorrhage were the predominating symptoms. Both patients were treated successfully by resection of the diverticulum.

PREOPERATIVE TREATMENT

Harrington stresses the desirability of clearing the sacculation of any foreign material which may be retained within it. This material may become so incrusted and thickened that removal may require repeated endoscopic aspirations. In neither of our cases was this technic utilized. Reliance was placed chiefly on the ordinary indications for surgical intervention including adequate fluid and blood replacement, administration of antibiotics, and the restoration to normal, or as nearly normal as possible, of the serum protein and electrolytic factors of the blood stream.

OPERATIVE TREATMENT

According to Harrington the separative procedure is resection of the diverticulum which, when it is of the epiphrenic type, includes chiefly esophageal mucosa. The diverticulum may be excised by removing the entire sac and retaining at least 1½ cm. of the mucosa beyond the true opening. At this point the diverticulum which has been excised is removed and the stump of mucosa inverted. The opening in the muscle wall is then closed with two rows of interrupted silk sutures. In a diverticulum of more moderate size the sacculation may be excised, the opening closed, and the wall of the esophagus plicated to normal size. In a small diverticulum the sacculation may be merely invaginated and the esophageal wall plicated to reinforce the new opening.

McNeally and Glassman[4] devised an ingenious method to overcome the esophageal symptoms of dysphagia and food retention due to a pulsion diverticulum of the esophagus in the epiphrenic site. They anastomosed the diverticulum to the stomach through the diaphragm, thus forming a new channel through which the diverticulum drained directly into the stomach,

FIGURE 1a *(Case 1)*. Preoperative roentgenologic findings. The thoracic esophageal diverticulum is located just above the diaphragm and takes its origin from the esophagus posteriorly and extends toward the right.

FIGURE 1b. Postoperative film, after excision of diverticulum.

without resecting the diverticulum itself. Most technics, however, consist of removal of the sacculation and repair of the defect in the esophageal wall. A somewhat different approach to the problem is illustrated by the first case here reported.

CASE REPORTS

Case 1—A 66 year old Negro was admitted to the Cook County Hospital with progressive weakness, anorexia and weight loss. The symptoms began some 12 years prior to admission with weakness, lassitude and inability to swallow. Ten years prior to admission he had a severe episode of weakness and lassitude and was treated at home. In August 1948 he became so weak that he was given three blood transfusions. He stated that he had lost between 50 and 60 pounds since the onset of the present illness. Intermittently he had dyspnea and palpitation of the heart with occasional edema of the extremities. Examination on admission revealed severe anemia with associated pallor and weakness. The anemia was so profound that a diagnosis of pernicious anemia or cancer of the gastrointestinal tract was considered. Blood pressure on admission was 100 systolic and 60 diastolic. The patient was moderately dyspneic. The remainder of the physical examination was negative.

Laboratory results—On admission the hemoglobin was 14 per cent with a red cell count of 1,680,000 per cubic millimeter. The color index was reduced to 0.42. The total proteins were 5.5 with the serum albumin 3.8 gm.; globulin of 1.7. The Kahn test was negative.

After repeated transfusions the hemoglobin rose to 80 per cent and the red cell count to

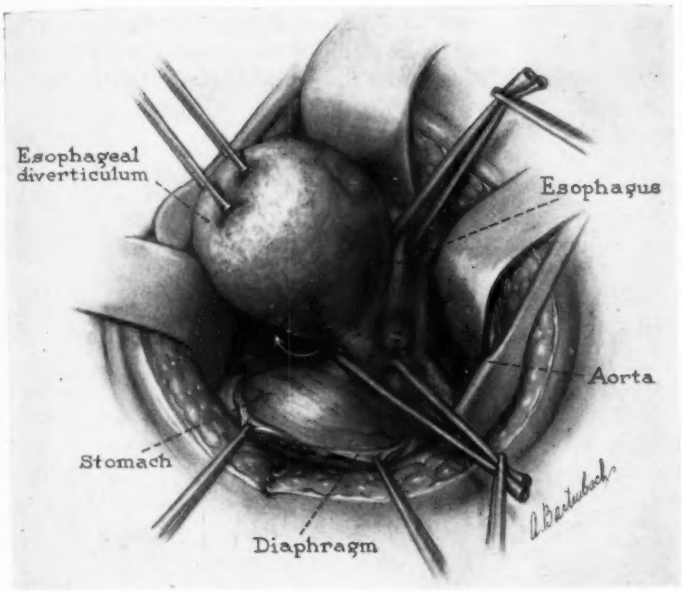

FIGURE 2. Technic of mobilizing stomach, esophagus and the diverticulum. Note again the proximity of the cardiac end of the stomach.

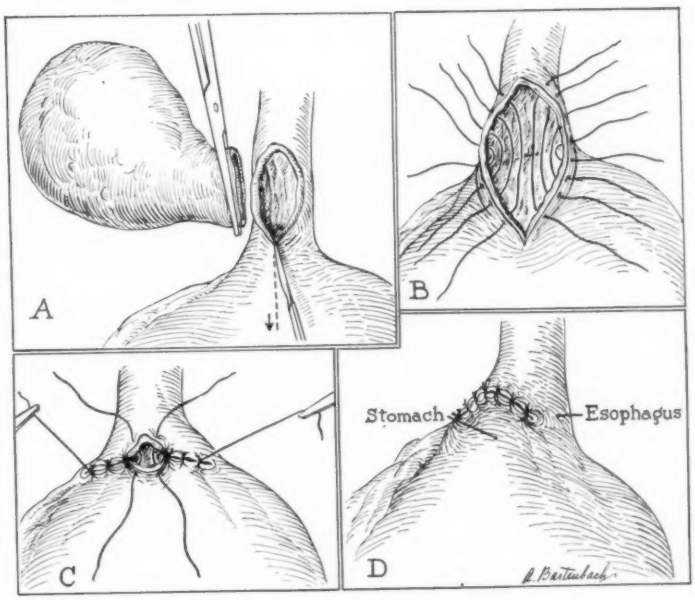

FIGURE 3. A, B, C and D. Various steps in the technic employed to repair the defect in the anterior wall of the esophagus resulting from excision of the diverticulum. Not illustrated are the further sutures used to reinforce the stoma by bringing the fundus of the stomach to the anterior wall of the esophagus, effectively inverting the first row of sutures and finally suturing the leaves of the diaphragm to the fundus of the stomach well beyond the stomal site to remove the tension from the gastroesophageal anastomosis.

4,880,000. Fluoroscopic examination with barium meal of the upper gastrointestinal tract showed a huge sac related to the upper end of the esophagus or the lower end of the stomach. Subsequent radiologic examination showed that this sac, which filled with barium, was related to the esophagus, and an esophageal diverticulum was suspected. However, because of its large size a gastric diverticulum could not be completely excluded. The diverticulum retained

at least one third of the barium substance in 24 hours.

Operation was performed under general endotracheal anesthesia. A posterolateral approach was made to the left thoracic cavity by resecting the tenth rib subperiosteally. When the inferior pulmonary ligament was divided, a huge sac was demonstrated, occupying most of the retrocardiac space and extending well in the direction of the right thoracic cavity from the lower portion of the esophagus. The diaphragm was opened and the stomach mobilized. The esophagus was then freed above and below the origin of the diverticulum. The diverticulum was dissected free from its bed and removed. Because of the extensive necrosis of the esophageal wall at the site of its origin, a portion of the esophageal lumen itself was resected with the mediastinal diverticulum. A longitudinal opening in the esophagus remained which was about 5 cm. long. A similar incision then was made into the anterior wall of the stomach and the opening subsequently was closed transversely after the technic of a Heineke-Mikulicz pyloroplasty. The fundus of the stomach was brought up over the suture line to cover the new esophageal gastric stoma which had been formed. Interrupted silk sutures were used throughout.

A subserous fibroma was found incidentally on the anterior wall of the stomach and was excised. The diaphragm was closed about the fundus of the stomach and the thoracostomy wound closed. A catheter was inserted through a separate opening for underwater drainage and the lung expanded. The patient made an uneventful recovery; liquids were allowed on the fifth postoperative day and solid foods shortly thereafter. He did not complain of dysphagia and began to gain weight almost immediately after he began eating. He left the hospital 25 days after operation in apparent good health.

Diverticula of the esophagus of the traction type seldom cause symptoms. They have but rarely been subjected to surgical removal.

Case 2—Mrs. R., 67 years old, was admitted to Woodlawn Hospital on February 16, 1949. Two weeks prior to admission she had had an attack of gross hematemesis, vomiting a large quantity of bright red blood. Bloody emesis persisted for the next 48 hours, gradually diminishing during this period of time. For the previous few weeks she had had questionable dysphagia. There were no other symptoms.

Roentgenologic examination of the entire gastrointestinal tract did not reveal a possible source of hemorrhage, but did show a small diverticulum in the midthoracic esophagus approximately 7 cm. below the superior border of the aortic arch. It was best viewed through the left anterior oblique exposure (Figure 4).

Laboratory results—There was a moderate anemia; the red cell count was 3,750,000 per cubic millimeter of blood, the hemoglobin concentration 11.9 gm. Electrocardiographic changes indicated mild changes compatible with myocardial damage. The interpretation was that the changes could be due to a reaction from the diverticulum.

The anemia was corrected by blood transfusions. On February 23, 1949, thoracotomy was performed. The left sixth rib was excised through a posterolateral incision. The pleural cavity was entered. The left lung was adherent to the parietal pleura with numerous filmy adhesions which were separated by blunt dissection. The inferior pulmonary ligament was exposed and divided, and the esophagus mobilized. A small diverticulum arising from the anterior wall of the esophagus was located. Its periphery just below the aortic arch was attached to a lymph node in the hilum near the bifurcation of the trachea. The lymph node and the diverticulum were dissected free. The stalk, consisting chiefly of the mucosa of the esophagus, was about the size of a pencil in diameter. The diverticulum was transected at its stump and removed together with the lymph node. The stump was inverted by a purse-string suture of silk, and the esophageal muscularis was approximated over this area with three interrupted silk sutures and reinforced further by approximation of some of the parietal pleura and a small wedge of lung tissue. The wound was closed in the usual manner and a catheter, inserted through a stab wound in the left ninth intercostal space, was brought out and placed under a water seal for 48 hours. There were no

FIGURE 4 *(Case 2)*. Preoperative esophagram, illustrating midthoracic diverticulum.

FIGURE 5 *(Case 2)*. Postoperative esophagram. Note the slight bulge of the wall of the esophagus at the site of excision of the diverticulum.

postoperative complications and the patient made an uneventful recovery.

The pathologist's report stated that the diverticulum was lined with squamous cell tissue typical of the esophagus. There was little or no muscularis tissue in the diverticulum. The lymph node presented chronic inflammatory changes. There were relatively few changes in the electrocardiographic tracings taken postoperatively. Subsequent examination of the esophagus revealed the lumen to be adequate with a slight bulge at the site of the removal of the diverticulum (Figure 5). The patient has remained in good health during the past three years and has had no further episodes of bleeding and no difficulty in swallowing.

SUMMARY AND CONCLUSION

Two instances of bleeding from thoracic esophageal diverticula are reported. A large pulsion diverticulum of the esophagus located in the epiphrenic area gave rise to repeated episodes of bleeding with chronic anemia and debility. In another instance a small traction diverticulum of the midthoracic esophagus caused massive bleeding. Both patients were apparently cured by surgical removal of the diverticulum.

Thoracic diverticula of the esophagus must be considered in the differential diagnosis of gastrointestinal bleeding. Surgical removal is feasible.

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Surgical Principles Involved in the Clinical Use of Streptokinase and Streptodornase

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Streptokinase and streptodornase, as adjuncts to surgical intervention, offer the surgeon a new biologic approach to the treatment of certain infections. The compounds are tools for the removal of fibrin and purulent exudates which enhance chemotherapeutic action and the healing of wounds.

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TILLETT and his associates[1, 2, 5-9, 12, 14] have demonstrated that the catalytic agent (streptokinase) and an enzymatic complex (streptodornase), which are produced in the growth of certain strains of beta hemolytic streptococci (group A), may be successfully employed in the topical treatment of certain acute and chronic infectious processes. Three years ago Tillett and Sherry reported on the beneficial effects of these compounds in the treatment of fibrinous, purulent and sanguineous pleural effusions.[10] More recently Tillett and his associates discussed their use as topical therapeutic agents in a number of infectious processes.[13] These excellent results have been confirmed by the authors of this report and by others.[3]

Streptokinase is a catalytic agent which activates a fibrin-lysing system present in the euglobulin fraction of blood. This causes liquefaction of fibrin. Streptokinase also inhibits the conversion of fibrinogen to fibrin. It is antigenic, and an antikinase may develop in human beings as the result of its use or as the result of a recent streptococcal infection.

Streptodornase is an enzymatic complex which hydrolyzes desoxyribose nucleic acid and desoxyribonucleoprotein. It requires catalytic activation by the magnesium or manganese ion. The phagocytic activity of polymorphonuclear leukocytes in infected material has been shown to be considerably enhanced by the depolymerization of desoxyribose nucleic acid and desoxyribonucleoprotein. The clumping of the leukocytes and consequent phagocytic inactivity are thus reduced to a minimum. The percentage of motile active forms is definitely increased locally. This

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TABLE 1

PRINCIPLES OF SURGICAL USE OF STREPTOKINASE AND STREPTODORNASE

1. Adequate provision for drainage of pus must be provided by using established surgical methods. The enzymatic compounds will promote drainage by thinning thick, purulent material.
2. Slough must be removed surgically from the wound since streptokinase and streptodornase will not dissolve it.
3. Foreign bodies must be removed from the wound.
4. Blood supply to the area must be adequate to provide the fibrin-lysing system and magnesium ions needed for enzymatic activity.
5. The enzymes must be placed in direct contact with their substrates in the area under treatment.
6. Products of digestion must be removed frequently and effectively.
7. Treatment should be on a continuous rather than an intermittent basis.
8. Systemic use of indicated chemotherapeutic or antibiotic agents is permissible.
9. Utilization of streptokinase and streptodornase permits the performance of a wide range of surgical procedures within a relatively short period of time. Planned cosmetic results may be obtained.
10. Use of the enzymes is not limited to the preoperative period.

is important, since phagocytosis is dependent on the presence of living motile leukocytes.

The local use of streptokinase and streptodornase offers the surgeon a new and efficient biologic approach to the treatment of certain types of infection. These compounds are, however, only adjuncts in the over-all treatment of such lesions. They are only a part of the approach to surgical treatment and, without competent surgical judgment and the utilization of proper technics, their use will often prove disappointing. When sound surgical judgment and principles are employed, streptokinase and streptodornase form valuable additions to surgical treatment.

The principles of the surgical use of streptokinase and streptodornase are summarized in Table 1. The fundamental principle is their contact with the substrates with which they will react. This often entails considerable thought and ingenuity. The use of catheters is an efficient method of accomplishing this in a deep wound. Superficial wounds may be treated directly with the solution. An alternative method proposed by Tillett and Sherry[11] requires the incorporation of the compounds in LUBAFAX, with 100,000 units of streptokinase and 60,000 units of streptodornase being suspended in 10 cc. of this water-soluble base. The addition of 250 to 500 mg. of crystalline aureomycin to this suspension might help control surface infection. Advantages of such a mixture are readily appreciated in treatment of superficial infections or burns. This method has been used successfully by the authors in a number of instances.

We feel that it is particularly important to remove the products of digestion from deep recesses of infected wounds, since it is thought that these products inhibit normal reparative processes. In addition, living leukocytes will not function in a pool of exudate. To render effective phagocytic action, leukocytes must move over the surface of a wound by ameboid action. In this state, they are most capable of phagocytosing offending micro-organisms.

Catheters, used as suggested for introducing streptokinase and streptodornase into the recesses of lesions, serve a double purpose. Streptokinase and streptodornase may be introduced into the wound to the desired point, and the leukocytic inhibiting products of depolymerization may be removed by air vent suction. Local collections of serum and blood also may be removed after plastic reconstruction of wounds, since the continued presence of such collections produces a dead space, prevents the apposition of skin flaps to underlying areas, and inhibits healing. Local collections of serum and blood make good culture mediums, and their removal will make bacterial growth more difficult.

THE best results in treatment of infected wounds with streptokinase and streptodornase have been achieved where complete exteriorization of the wound has been possible, since maximal action of the enzymes is produced only when and where adequate drainage is present. Localizations of purulent material must be opened. While it is true that purulent secretion will be thinned by the enzymes in sites with inadequate drainage and that a greater amount of thinner secretion will result, the desirable maximal action is not obtained. Slough which prevents satisfactory contact of the en-

FIGURE 1 *(Case 2)*. Wound on October 18, 1950, just before treatment with streptokinase and streptodornase was started.

FIGURE 2 *(Case 2)*. Wound appears much smaller and cleaner on October 27, 1950.

zymes with constituents of the wound to be hydrolyzed must be removed from infected wounds to obtain maximal enzymatic action. Slough may also contain trypsin, which will inactivate streptokinase.

The release of purulent material also permits the more effective action of immune bodies, the sulfonamides, or antibiotics, if the latter are being administered. Foreign bodies which hamper action of the enzymes also must be removed.

The blood supply to the local area must be good, or efficient action of the enzymes will not be obtained. The fibrin-lysing system which is in the euglobulin fraction of the plasma must be carried to the site of the lesion, since streptokinase per se does not possess the ability to lyse fibrin. Likewise, since the magnesium or manganese ion is necessary for activation of streptodornase, a good blood supply is necessary to provide these activators. Improvement of the local blood supply may be indicated, either by application of heat, by elevation of the affected part to decrease edema, by blocking the regional portion of the sympathetic nervous system with procaine, or by performing sympathectomy.

In sterile or infected hemothoraces, or in cases of empyema, 300,000 units of streptokinase and 200,000 units of streptodornase are dissolved in 20 cc. of sterile isotonic solution of sodium chloride. This solution is injected slowly and gently into the affected pleural cavity through

FIGURE 3 *(Case 2)*. Wound on November 8, 1950, showing continued improvement.

FIGURE 4 *(Case 2)*. Wound on February 23, 1951, after completion of the transfer of the cross leg flap.

a previously placed intercostal catheter provided with under-water drainage. Eight hours later the products of hydrolysis are removed by applying a small amount of suction (–6 to –8 cm. of water) to the system by means of a Stedman pump. Then a fresh solution of streptokinase and streptodornase is injected and the procedure is repeated until the pleural cavity is believed to be clear.

For infected superficial and deep soft-tissue wounds, 300,000 units of streptokinase and 200,000 units of streptodornase are dissolved, with minimal agitation, in 20 cc. of sterile saline solution. This solution is applied topically to the wound, which is then covered with NYLON® gauze. If the wound is deep, catheters are placed in the wound, a piece of nylon gauze invaginated into the area over the catheters, and the area packed with ordinary gauze. The enzyme solution is injected slowly and easily through the catheters.

Lubafax has proved to be a useful vehicle for streptokinase and streptodornase in treatment of superficial infected wounds and burns. Crystalline aureomycin to control surface infection can be incorporated into the suspension with advantage. As currently used, 300,000 units of streptokinase, 100,000 units of streptodornase and 0.5 gm. of crystalline aureomycin are dissolved in 5 cc. of physiologic saline solution. The resulting solution is mixed with 30 cc. of lubafax, which is a water-soluble jelly. The mixture is stored in the refrigerator and used as needed. It will retain its potency for at least one week.

It was apparent that our results in osteomyelitis might be improved by the use of 1:1000 aqueous ZEPHIRAN CHLORIDE® as a cleansing solvent, and also by the use of a small amount of dissolved aureomycin to control surface infection. Aureomycin is preferred to penicillin and other antibiotics which not infrequently produce sensitization when used locally, since in our experience sensitization to aureomycin is rare. Neither an aqueous form of zephiran chloride nor a suspension of aureomycin seems to inhibit the catalytic action of streptokinase on the fibrinolysin system in plasma clot experiments made in vitro.

Another addition to the chemical treatment of osteomyelitis has been the use of a 1 per cent solution of acetic acid in glycerin. Acetic acid will inhibit the conversion of trypsinogen to trypsin, and also will inactivate trypsin present locally in a wound. Trypsin will hydrolyze streptokinase. Glycerin will lower the local surface tension and produce greater penetrating power for the acetic acid in areas in which bone is present. The empirical use of acetic acid as an adjunct to streptokinase and streptodornase in the chemical debridement of wounds has been confirmed in the laboratory by Sherry.[4] Collagen is not affected by streptokinase and streptodornase, but it is dissolved by dilute acetic acid.

THE present method of treating osteomyelitis is, therefore, a planned approach using a combination of surgical and chemical debridement. Details of the enzymatic and chemical treatment may be summarized briefly: 300,000 units of streptokinase and 200,000 units of streptodornase are dissolved in 20 cc. of a 1:1000 solution of aqueous zephiran chloride, and 250 to 500 mg. of aureomycin hydrochloride are dissolved in this solution. Use of the sterile buffered solution of aureomycin may be restricted to the serous cavities. A round-the-clock schedule of treatment is suggested. At 8:00 A.M., 4 cc. of a 1 per cent solution of acetic acid in glycerin is injected into the infected area through a segment of a No. 6 French ureteral catheter or a piece of small-bore polyethylene tubing. At 12:00 noon, the wound is irrigated with normal saline solution. Four cubic centimeters of the solution of streptokinase-streptodornase-aureomycin are injected immediately after the saline wash at 12:00 noon and again at 4:00 P.M., 8:00 P.M., 12:00 midnight and 4:00 A.M. At 8:00 A.M. the following day the wound is washed with normal saline solution and the cycle is repeated. The acetic acid may be used more frequently if the condition of the wound demands it, in which case it is substituted for one of the applications of streptokinase and streptodornase.

Other types of infection may demand the use of other antibiotics. Recently a severe ocular infection due to *Bacillus pyocyaneus* was successfully treated by the systemic and local use of polymyxin B in combination with a saline solution of streptokinase and streptodornase. Strepto-

kinase is not inactivated by polymyxin. The infected eye was washed at four hour intervals with a saline solution of polymyxin B, which was alternated with four hour applications of streptokinase and streptodornase.

The type of organism present does not seem to influence the result of treatment, since infections due to all types of bacteria, including gas-forming anaerobes, tuberculi, and infection due to *Nocardia,* have been successfully treated. Fibrin, desoxyribose nucleic acid and desoxyribonucleoprotein are found in the exudate from all these infections, and streptokinase and streptodornase are effective in ridding the wounds of these materials. In superficial wounds, in which infected collagen is a factor in slowing the healing process, acetic acid will hydrolyze this substance.

The toxic effects from the topical application of streptokinase and streptodornase are practically negligible. Use of the compounds in closed spaces, such as the pleural cavity and knee joint, will produce a moderately high elevation of temperature which may last 12 hours or more. The products of degradation probably are a factor in producing this elevation. A second possible factor in producing a pyrogenic response is a direct result of the cleansing produced by these compounds. The lysis of fibrin and desoxyribonucleoprotein from the walls of a closed space will release bacteria which have been imprisoned, and it is not uncommon to obtain positive cultures after several days of treatment even though previous cultures had been sterile. Minor headaches and aching have been seen more frequently when the compounds were used in closed spaces.

Prothrombin times which have been calculated for patients treated with these enzymes have not shown a significant deviation from the normal. Hematomas and areas of ecchymosis have not been seen. Inadvertent bleeding has not been produced in the treatment of hemothoraces or in the immediate postoperative application of the compounds to reconstructed wounds. In the latter instances, it is believed that the thrombus in individual vessels is of great enough length so that the lysing action produced in the more superficial area is not of consequence.

CASE REPORTS

Case 1—A 31 year old Negro was admitted to the Veterans Administration Hospital in Fort Howard, Maryland, on May 9, 1950, for treatment of a painful mass about 3.5 cm. in diameter in the right supraclavicular region. This mass had been present for about five weeks and had grown progressively larger. Aspiration on May 12 showed acid-fast bacilli. Aerobic and anaerobic cultures were sterile. Streptomycin (0.5 gm.) was given intramuscularly from May 16 through May 22. Incision for drainage was done on May 19, and 80,000 units of streptokinase and 38,000 units of streptodornase were applied topically to the wound four times between May 20 and May 26. Closure of the wound was done on May 29. A slight amount of purulent drainage, from which hemolytic *Staphylococcus aureus* was isolated, was noted on June 3. The wound was healed on June 15, and the patient was discharged on June 23. He was re-admitted to the hospital March 1, 1951, for treatment of an unrelated disease. The tuberculous lymphadenitis had not recurred.

Case 2—A 27 year old white man was admitted to the hospital on October 16, 1950. He had been injured by a shotgun wound in the inner aspect of the left foot on September 15, and had been treated elsewhere with debridement, cast and penicillin. Examination revealed a necrotic wound, 6 by 4 by 1.5 cm. in size, in which several tarsal bones were exposed. *Staph. aureus* was cultured from the wound. Treatment consisted of a routine of acetic acid, zephiran chloride, aureomycin and streptokinase and streptodornase on a four hour schedule, the solution being introduced through a short segment of ureteral catheter. The solution used consisted of 200,000 units of streptokinase and 300,000 units of streptodornase dissolved in 20 cc. of 1:1000 aqueous zephiran chloride, to which 250 mg. of aureomycin was added. At 8:00 A.M., 4 cc. of 1 per cent acetic acid in glycerin was introduced into the wound. At 12:00 noon, the wound was washed with physiologic saline solution, and 4 cc. of the streptokinase-streptodornase solution was injected. These 4 cc. injections were repeated at 4:00 P.M., 8:00 P.M., 12:00 A.M. and 4:00 A.M. The entire procedure was

FIGURE 5 *(Case 3)*. Empyema on April 5, 1951, before treatment.

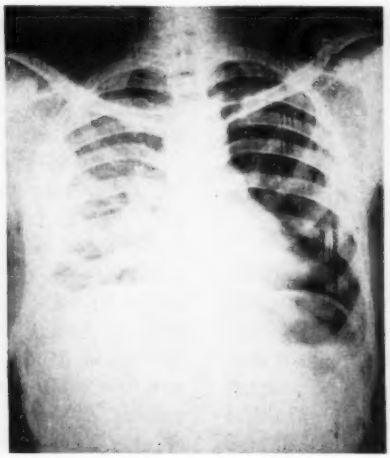

FIGURE 6 *(Case 3)*. Termination of treatment with streptokinase and streptodornase before partial withdrawal of the catheter on April 12, 1951.

FIGURE 7 *(Case 3)*. Day before discharge, April 23, 1951.

again started at 8:00 A.M. This routine was continued from October 18 (Figures 1, 2 and 3) through November 23, when the wound was quite clean.

A cross leg flap was started on October 19, three days after the patient was admitted to the hospital. Topical applications of 40,000 units of streptokinase and 60,000 units of streptodornase were given from November 25 through December 12. On December 13 the dose was reduced to 12,000 units of streptokinase and 10,000 units of streptodornase; this was continued until January 17, 1951, when the flap—which had undergone multiple preparatory stages—was transferred. Partial transection of the flap was done on February 3 and on February 5 and it was completed on February 7. The flap was attached to the posterior part of the wound on February 15 (Figure 4). Three hundred thousand units of crystalline procaine penicillin G in oil was given from December 7 through December 12. The patient was discharged on March 3, 1951.

Case 3—A 24 year old Negro was admitted to the medical service of the Veterans Administration, Fort Howard, Maryland, on March 5, 1951, with bilateral pneumonia. He appeared acutely ill. Culture of the sputum showed pneumococci and culture of the blood revealed about 500 colonies of pneumococci per cubic centimeter. One hundred thousand units of crystalline procaine penicillin G was given intramuscularly every three hours from March 6 to March 11; then the amount was increased to 150,000 units and continued to March 20. Four grams of sulfadiazine was given intravenously on March 6, followed by 1 gm. orally every four hours through March 16. The response to treatment was excellent. Culture of the blood on March 12 was sterile, both aerobically and anaerobically.

On April 5 a right empyema developed (Figure 5) and the patient was seen in consultation. Intercostal closed drainage was established on the same day. Suction in the system of about 6 cm. of water was obtained by use of a Stedman pump. Culture of the purulent fluid was sterile aerobically and anaerobically. A solution of 150,000 units of streptokinase and 50,000 units of streptodornase was injected through the catheter daily from April 7 through April 11. The catheter was occluded after the injection of the compounds and digestion allowed to procede for eight hours. Suction drainage was reestablished after this period. Three hundred thousand units of crystalline procaine penicillin G in oil was given intramuscularly twice a day

from April 17 through April 23. Response to treatment was excellent (Figure 6). Suction on the catheter was discontinued, the catheter was partly withdrawn and simple under-water drainage established on April 12. The catheter was removed on April 17. The patient, recovered from his empyema (Figure 7), was discharged from the hospital on April 24.

Case 4—A 24 year old white woman was admitted to the U. S. Marine Hospital, Baltimore, Maryland, on February 8, 1950, with fever, nausea, vomiting and abdominal pain. Treatment with antibiotics was started and continued for 35 days. A mass in the right lower quadrant developed. When this mass was incised, it was found to contain a large amount of purulent material from which a strain of Bacteroides was obtained. For the next 70 days the patient was extremely ill, with elevations of temperature, at times, to 106° F. The development of a subphrenic abscess in the cul-de-sac necessitated incision to provide drainage. Penicillin, sulfonamides, aureomycin and bacitracin were given in large amounts. Pyelophlebitis, ascites and jaundice developed and cleared. The patient was discharged in July 1950. An appendectomy was done in September 1950 without incident.

In October 1950, the patient was again admitted in a toxic condition with a large mass in the right abdominal quadrant. Incision and provision of drainage released only a small amount of purulent material. The wound healed but another mass developed in the same area in November 1950. Incision and drainage again produced a small amount of purulent material. The wall of the mass was estimated to be about 10 cm. thick. Repeated cultures showed Bacteroides. A sump drain was introduced into the tract on November 25, 1950. A solution, comprising 100,000 units of streptokinase and 60,000 units of streptodornase in 100 cc. of physiologic saline, was divided into six parts and one part was injected into the sinus tract while the suction on the sump drain was discontinued. Digestion was permitted for two hours, after which the suction was started for one hour. The cycle was then started again and continued during the day until six doses had been given. Treatment was continued through December 4. The patient was discharged on December 19. On a return visit on January 25, 1951, the patient stated that she had remained well. The abdominal wound was well healed and there was no evidence of a mass in the abdomen.

Case 5—A 22 year old white man was admitted to the U. S. Marine Hospital on August 28, 1950, for treatment of a swollen, hot, painful left knee. The patient had been injured in 1949 and arthrotomy for a lateral meniscectomy had been done twice at another hospital. A synovectomy was done in September 1950 and postoperative aspiration of the joint was required a number of times. The patient left the hospital on October 20. He returned on October 26 when the knee was painful, swollen and hot. Aspiration on the same day produced 49 cc. of thick yellow fluid from which hemolytic *Staph. aureus* was cultured. The knee joint was explored on November 17 and found to be filled with an amorphous semifluid material which, on microscopic study, was found to be almost pure fibrin.

A solution of 100,000 units of streptokinase and 60,000 units of streptodornase was injected into the joint on December 5. This produced an immediate increase in pain, more severe swelling and an elevation of temperature to 102.8° F. On December 6, 100 cc. of thick creamy material was aspirated from the joint and the same amount of the drugs was injected. A sinus tract developed the following day and an unknown amount of drainage was noted on the dressings. The patient was seen in consultation on December 9 when polyethylene tubes were placed into the joint from the medial and lateral aspects. A solution of 200,000 units of streptokinase and 300,000 units of streptodornase dissolved in 10 cc. of physiologic saline was injected through one of the catheters daily. After four hours of digestion, suction was applied to one of the catheters. Treatment was continued daily through December 13. A large amount of drainage was noted on that day. The patient was discharged on March 20, 1951. Since that time, a slow and steady recession of the pain and swelling has occurred. A slight thickening of the periarticular tissues has persisted but motion is normal except for a slight loss of extension.

Case 6—A 70 year old white man was admitted to the Maryland General Hospital on December 13, 1950, with a laceration of the left forearm incurred from a buzz saw. The laceration was repaired the same day. An infection developed, from which *Clostridium welchii* was isolated. The wound was opened widely and 300,000 units of streptokinase and 180,000 units of streptodornase were applied topically on December 24. The following day, 200,000 units of streptokinase and 120,000 units of streptodornase were applied in a similar manner. The patient was seen in consultation on December 28. One hundred thousand units of streptokinase and 60,000 units of streptodornase were dissolved in 20 cc. of physiologic saline. A plastic catheter with a small base was placed at each end of the wound, and the solution was divided into four parts and injected into the wound at intervals of six hours. Treatment was continued through January 4, 1951. The patient was given penicillin and aureomycin. Cultures of the wound on January 3, 5 and 8 showed only nonhemolytic *Staph. aureus*. A full-thickness flap was transferred to the left arm from the abdomen on January 10. The transfer was completed on February 1. The wound healed and the patient was discharged from the hospital on February 7.

COMMENT

A successful result will not follow the use of streptokinase and streptodornase in all instances of infected wounds. However, because the results of treatment have been uniformly good, a careful estimate of the situation must be made when effective action of the compounds is not obtained.

Care in storage of the desiccated form must be exercised, and the ampules should be kept at a temperature of 4° C. When a solution of streptokinase and streptodornase has been prepared, it should be stored in a refrigerator and used within 24 hours.

Violations of sound surgical principles when these compounds are used will frequently produce poor results. These enzymes are at best only a local tool, and their use must be integrated in a rational plan of treatment if maximal results are to be obtained.

The compounds must be in direct contact with the area in which their action is desired.

The products of digestion must be removed. Accumulations of exudate will delay the sterilization and healing of the wound.

Streptokinase and streptodornase are not specific curative agents. Rather, as a result of their ability to liquefy fibrin, desoxyribonucleoprotein and desoxyribose nucleic acid, they are cleansing in their action, thus permitting the more effective use of chemotherapeutic and antibiotic agents, and creating conditions which promote wound healing. Proper use of these enzymatic compounds will result in a saving of time in wound repair.

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Use of Cortisone and Antibiotics in Resistant Pelvic Infections

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Although the incidence of resistant pelvic infections has been decreased, they are still a problem in spite of adequate antibiotic therapy. A new approach to such cases, in the subacute stage, is described, and the method, rationale and dangers outlined. Results obtained in the cases described, as well as in others treated subsequently, have been excellent.

Since the advent of the various antibiotics, pelvic infections have not been the killing, crippling and sterilizing diseases they were 10 years ago. The incidence of the "frozen pelvis" has decreased dramatically. On our Public Service we attribute the decreased mortality and morbidity rates to the following factors: (1) early and massive antibiotic therapy; (2) no surgical interference except in the presence of abscess formation; (3) intensive supportive measures including frequent blood transfusions and heavy protein feedings; (4) earliest possible ambulation, and (5) initiation of treatment early in the course of the disease.

All pelvic infections (except gonorrheal) are treated by us as potential peritonitis cases and we depend on the injection of 100,000 units of aqueous intramuscular crystalline penicillin every three hours, as first recommended by Crile. At the same time, without waiting for identification of bacteria, we administer streptomycin, 0.5 gm., intramuscularly every six hours. Of late we have been substituting aureomycin in doses of 500 to 750 mg. every six hours for both penicillin and streptomycin. The longer the disease has prevailed before admission of the patient, the slower the response, even to increased doses of antibiotics, i.e., the more resistant it is to treatment. Our trials with procaine penicillin did not give us nearly as good response as did the regimen already described, even though the total number of units employed was the same. A review of our last 75 cases (excluding gonorrheal infections) reveals a mortality of zero, three abscesses in the pouch of Douglas and only three "frozen pelves"—one of which was a puerperal exacerbation of a tuberculous pelvic peritonitis and will not be considered further.

In the past the "frozen pelvis" usually has referred to residual infection and inflammation which remain long after the initial fulminating and possibly killing peritonitis, parametritis and perimetritis have subsided. The patient becomes a "pelvic cripple." Her hospitalization is prolonged, her convalescence unending. She suffers from menorrhagia, metrorrhagia, polymenorrhea, chronic pelvic pain and severe dysmenorrhea. She cannot do work of any kind. She becomes anemic and nervous and, after endless therapy, including all forms of diathermy,

the inflammatory reaction in the pelvis disappears, leaving a mass of scar tissue. Then hysterectomy is performed. This, however, cannot be done until "the infection has subsided." Previously, surgeons have always waited two to three years before feeling that it was safe to reinvade the infected area. How long the waiting period is at present, with the aid of antibiotics, I do not know. After the waiting period, of course, the pelvic organs are a solid mass of distorted scar tissue—from the surgeon's viewpoint. The pathologists, however, always comment on how few changes they find inside the uterus, tubes and ovaries and how resistant they are to the infection which was present.

In this paper we are interested in the subacute phase of the process described—the period when the acute fulminating process has subsided and there remains in the pelvis a mass of infected granulation tissue which will respond neither to antibiotic therapy nor to physiotherapy. At this stage the body has walled off the infection, not into one large abscess which can be approached surgically, but into many tiny ones. The patient bleeds; pus and blood are discharged from the uterus; she runs a low grade fever. She has constant pain, especially on movement. She is waiting for the inevitable hard scar tissue which will protect her from the disease, but will also cripple her with permanent pain. No surgeon who has inadvertently invaded one of these pelves at this stage wants to repeat the procedure.

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THE THEORY

We know that the ability of antibiotics to destroy susceptible invading organisms depends on the plasma concentration of the antibiotics. We know, for instance, that streptomycin will kill tubercle bacilli in the test tube and that it is also effective where the bacteria are free in the body, i.e., miliary tuberculosis, including tuberculous meningitis. We know that it is ineffective in calcified, chronic tuberculous lesions. It would seem logical that, in granulomatous lesions, not only has the body walled off the invading organism from itself with scar, hyalin, collagen or calcium, but it also has built a defense for those organisms against attack by any bacterial antagonist the body can muster, i.e., opsonins, agglutinins and leukocytes, or agents which the body can accept artificially, such as antibiotics. Therefore, if the bacteria are not killed early in the course of the disease, they become resistant—not in themselves, but because they themselves lie safely buried inside the granulation tissue or granuloma laid down. Hence flare-ups occur in surgery on old osteomyelitis cases or peritonitis in old P. I. D.'s, etc.

Selye has demonstrated repeatedly the ability of cortisone to dissolve recently laid-down granulation tissue. Brown has stated repeatedly that cortisone must not be administered in cases of pulmonary tuberculosis because of the danger of widespread dissemination and death. Selye has shown that healthy animals can be killed by overdosage with cortisone because it lowers tissue barriers and allows normally nonpathogenic bacteria to invade.

If we are sure of the specific bactericidal capacity of an antibiotic, would it not seem logical to blast away the walls of granulation tissue with cortisone and then let the increased local plasma concentrations of antibiotic sweep

up like the infantry after an artillery barrage? It is to be understood, of course, that the use of cortisone alone would be dangerous in such cases and quite possibly fatal.

CRITERIA FOR THE STUDY

It was decided that no patient would be treated in the manner described unless she fulfilled certain criteria:

1. The patient must have resisted response to standard methods of treatment over a lengthy period.
2. The acute phase of the disease must have passed and the patient inevitably be faced with a lengthy crippling illness.
3. The patient must be essentially stable.
4. The patient must be seen in consultation by a qualified internist before and during the therapy.
5. The patient must be seen in consultation by the chief of gynecology, and the chronicity of the disease established and therapy approved.
6. Adequate laboratory controls must be furnished by the Department of Pathology.
7. The patient's present status and possible dangers of the treatment contemplated must be explained to the patient.

CASE REPORTS

Case 1—A 21 year old unmarried woman, para 2, was admitted in December 1950 after having been confined at home for nine days with an infected, incomplete abortion, generalized peritonitis, parametritis, metritis and endometritis. She was extremely ill on admission. A pure culture of nonhemolytic *Bacillus coli* was isolated in the sanguineous, purulent discharge from the cervix. A roentgenogram showed a diffuse, indefinite mass filling the pelvis. The Aschheim-Zondek test was positive. She received 100,000 units of penicillin every three hours, streptomycin, 0.5 gm. every six hours—to which aureomycin, 750 mg. every six hours, was added one week later—and multiple blood transfusions and routine therapy. On the sixteenth day a large abscess, localized in the pouch of Douglas, was easily and adequately drained. The patient was relieved of pain but, despite continuous antibiotic therapy in the described doses and application of heat to the pelvis, she continued to have a daily temperature of about 100° F., continued to bleed, had severe pain on movement and, despite heavy protein and vitamin feedings and multiple blood transfusions, her condition continued to decline.

On the forty-first day of her hospitalization, her temperature was 100° F., her eosinophil count was 119, and her sedimentation rate was 48. The uterus and cervix were immobile, the parametrium was extremely hard, and there was pelvic tenderness (Figure 1). Vaginal bleeding required the use of two sanitary napkins daily. At this time—25 days after drainage of the abscess—treatment with cortisone was started. Penicillin, streptomycin and blood transfusions were continued as needed. Aureomycin was discontinued.

On the third day of cortisone treatment, the uterus and cervix were slightly mobile, there was less hardness of the parametrium and less pelvic tenderness, and the vaginal bleeding required the use of only one sanitary napkin daily. The sedimentation rate had increased to 58 and the eosinophil count to 212. On the sixth day of

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FIGURE 1. Forty-first day of disease and twenty-fifth postoperative day.

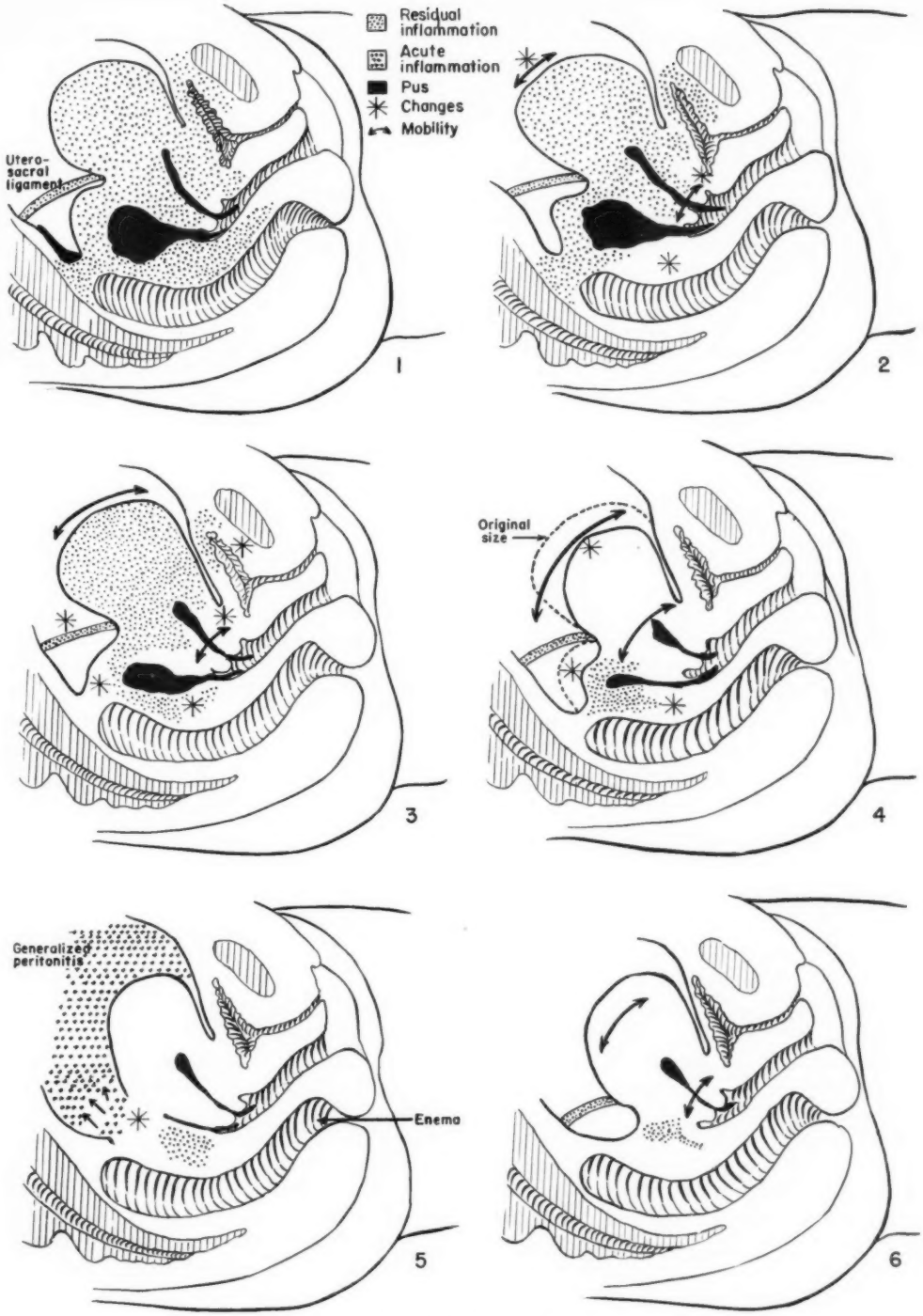
FIGURE 2. Fixed. "Frozen."

FIGURE 3. Forty-fourth day of disease; twenty-eighth postoperative day and third day of treatment with cortisone.

FIGURE 4. Forty-seventh day of disease; thirty-first postoperative day and sixth day of treatment with cortisone.

FIGURE 5. Forty-ninth day of disease; thirty-third postoperative day and eighth day of treatment with cortisone.

FIGURE 6. Fiftieth day of disease; thirty-fifth postoperative day and tenth day of treatment with cortisone.



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cortisone treatment, the temperature was 98.4° F., sedimentation rate 24, eosinophil count 175. There was no vaginal bleeding, only slight pelvic tenderness, and mobility of the uterus and cervix was considered moderate. These pelvic findings were confirmed by myself and others of the teaching, resident and intern staffs. There was a dramatic change in the patient as well as in her pelvic condition. The typical cortisone euphoria appeared but, at the same time, temperature became normal and remained so for the first time in 47 days and the pelvis softened and became mobile and nontender.

On the tenth day of cortisone treatment, a dramatic episode occurred. Due to factors beyond our control we could obtain no more cortisone for about 60 hours. Despite increased doses of antibiotics, blood transfusions, oxygen, etc., the patient's temperature rose to 105° F., her pulse rate to 140, and she went into shock and became comatose. Her condition was much worse, in fact, than it had been on the day she was admitted to the hospital. I am quite sure that without cortisone she would have died. As soon as a new supply of the drug was obtained, the patient's condition improved in a period of hours and returned to its former level. The second course of cortisone lasted for eight days, after which time her temperature became normal and remained so, her sedimentation rate was 23, and eosinophil count 210. Twenty days after cortisone had been discontinued, normal menstruation occurred.

Administering cortisone in such a case is like holding a lion by its tail—it is impossible to let go. Therapy must be constant and it cannot be stopped until all bacteria present are destroyed. It is important to note, however, that although at this stage the patient had the same abdominal pathology as she had on admission, the same antibiotic therapy plus cortisone provided a complete local and general cure. Cortisone was given at a rate of 300 mg. the first day, 200 mg. the second day and 100 mg. each subsequent day but, because of inadequate eosinophil, sedimentation rate and temperature response, the amount was increased to 150 mg. per day and maintained at this amount. Cortisone was tapered off at the rate of 25 mg. per day when the pelvis was clear on physical examination and only then were the antibiotics gradually tapered off. The pelvis, temperature and sedimentation rate remained normal even when the eosinophil count returned to its normal rate. The patient was last seen in the Outpatient Department on July 15, 1951, five months after being discharged from the hospital. She was symptom-free, working regularly and having regular menstrual periods with no dysmenorrhea, menorrhagia or leukorrhea. She planned to be married shortly.

Case 2—A 31 year old married woman was admitted to the hospital complaining of severe lower abdominal pain, profuse sanguineous, purulent discharge, and fever and chills, all of two and one-half weeks' duration. One month before admission, she had had dilatation and curettage and cauterization of the cervix at another hospital. Physical examination showed a toxic, anemic and feverish woman. The parametrium was hard and tender; the cervix was fixed, tender, painful and closed. The uterus was in normal position but could not be outlined from a hard, tender, nonfluctuant mass which extended from the uterus to the sides of the pelvis and involved the bladder and urethra, to the pubic rami. Culture from the cervix showed a pure growth of hemolytic *B. coli*.

Treatment for one week with the previously described doses of penicillin and streptomycin was ineffective. The local condition was unchanged; dysuria was severe. Afternoon temperatures persisted at 100° to 101° F. Treatment with aureomycin, 750 mg. every six hours for the second week, was also ineffective.

One month after the onset of the patient's illness and two and one-half weeks after admission, cortisone treatment was started—300 mg. the first day, 200 mg. the second day, followed by a daily maintenance dose of 100 mg., together with the aforementioned schedule of aureomycin. Adequate supplies of cortisone were assured before we began therapy. Softening of the lesions was demonstrable within 48 hours. The uterus could be outlined for the first time on pelvic examination on the third day, and the temperature was normal for the first time since admission 17 days earlier. The para-

metrium softened at once and on the fourth day both hands could be brought together behind the pubis for the first time, purulent discharge from the cervix stopped, and the patient's principal complaint, dysuria, disappeared. Euphoria was so great that it was difficult to keep her in bed. Eosinophil response was adequate and sedimentation rate dropped from 76 to 8 in six days.

As soon as the pelvis was entirely soft—in eight days—the cortisone was tapered off at the rate of 25 mg. per day. On the day cortisone was discontinued, aureomycin was tapered off at the rate of 250 mg. per dose per day. After therapy was stopped, there was no recurrence of pain or elevated temperature. Sedimentation rate remained normal even when the eosinophil count returned to normal, and the pelvis retained its normal pliability and lack of tenderness. One week later the patient was discharged, asymptomatic. Two weeks later she was seen in the Outpatient Department, asymptomatic, with a normal pelvis.

SUMMARY

1. The problem of subacute and chronic infection in the human body is still present despite the efficacy of antibiotics against the bacteria involved. This efficacy seems to diminish with the increase in duration of the specific infection.

2. A theory is postulated to explain the inability of antibiotics to attack bacteria in granulating lesions and perhaps granulomas which have become subacute and which tend to become chronic.

3. The possible action of cortisone with antibiotics in such cases is hypothesized.

4. The dangers of such treatment must be fully appreciated. Once started, cortisone cannot be withdrawn until the patient is cured and all infecting organisms are destroyed.

5. Two case reports, showing identical responses, are presented.

6. The sedimentation rate and eosinophil count are excellent guides to the adequacy of cortisone dosage. Only when the sedimentation rate remains normal, when the eosinophil count has risen to normal after cortisone has been discontinued gradually, and when clinical findings show disappearance of the infection can antibiotic therapy be tapered off.

7. Because of the short term of cortisone therapy in such cases, elaborate blood chemistry and ketosteroid controls are not necessary. They were used in the first case and were of no help. They were not used in the second case.

8. It is possible that the same combined therapy might be used successfully elsewhere in the human body.

9. The same approach is too dangerous to use on tuberculous infections at present, until it is substantiated by adequate animal experimentation.

Acknowledgment is made to Dr. A. L. Richard, Dean of the Faculty of Medicine, University of Ottawa, who was consulted in all stages of the cases discussed and who gave constructive critical advice throughout; to Dr. A. Fidler, Professor of Medicine, who established the adequate criteria for treatment and control, and to Dr. D. Magner, Professor of Pathology, whose assistance was generous and time-consuming.

The cortisone used was financed through a grant by the Faculty of Medicine of the University of Ottawa. The aureomycin used for the second patient was supplied by Lederle Laboratories.

COMING MEETINGS AND POSTGRADUATE COURSES

University of Minnesota Medical School, Minneapolis

Course in pediatrics, April 21-23, at Center for Continuation Study, intended primarily for pediatricians. Emphasis will be placed on infectious diseases. Visiting faculty member will be Dr. John M. Adams, University of California, who will also deliver the Clarence M. Jackson Lecture on "Respiratory Diseases—Changing Concepts."

Continuation course in proctology, April 14-19, at the Center for Continuation Study. Intended primarily for general practitioners, the course will cover all aspects of anorectal disease and will include operative clinics. Visiting faculty member will be Dr. Garnet W. Ault, Washington, D. C.

1952 Industrial Health Conference

Convention to be held April 19-26 at Netherland Plaza Hotel, Cincinnati, Ohio.

University of Colorado School of Medicine, Denver

For further information on the course listed below, write to Postgraduate Education, University of Colorado School of Medicine, 4200 East Ninth Avenue, Denver, Colorado.

Gynecology, obstetrics and related problems of the newborn, April 10-12, highlighted by four prominent guest lecturers: Dr. Emil G. Holmstrom, University of Utah; Dr. William C. Keettel, University of Iowa; Dr. Gilbert J. Vosburgh, Western Reserve University, and one guest pediatrician.

Diagnostic Clinic

Postoperative Complications due to Electrolyte Disturbances

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THERE seems to be a great deal of confusion in the minds of surgeons and others as to the water and electrolyte needs of postoperative patients, probably because of the swing of the therapeutic pendulum during the last many years. For example, in the period dating from 1920, the need for administering sodium chloride became more and more apparent, and patients were given 3, 4 and 5 liters of isotonic saline solution per day. The beneficial effects observed in many of these patients were undoubtedly due to the correction of chloride deficits during or after operation, but its routine use when such deficits were not present led to fatalities.

By 1945 it became quite apparent that the routine administration of such large amounts of salt frequently led to postoperative difficulty, so that this erroneous form of therapy was called postoperative salt intolerance. However, the knowledge that salt was poorly tolerated by the postoperative patient had appeared in the French literature as early as 1903.

The gradual realization that too much salt was harmful was followed by the administration of less and less salt. The trend continued, so that at the present time many patients receive no salt at all, a common routine being the administration of 3 or 4 liters of glucose in water. As a result it is not uncommon to see postoperative patients with evidence of water intoxication. It is no wonder that many surgeons are confused and demand accurate information upon which they may base an adequate intake of salt and water after operation.

The problem was at first complicated and later clarified by the discovery that potassium losses may lead to severe clinical manifestations, so that potassium salts must be considered an essential part of the electrolyte intake.

First let us consider the effect of trauma or operation upon water and electrolyte metabolism (Figures 1 to 4).

In Figure 1 are data by Dr. C. A. Moyer comparing the effect of 3 liters of saline and 3 liters of glucose, first in a normal individual and then in a patient the first day after abdominoperineal resection. With 3 liters of sodium chloride given over a 24 hour period, the plasma chloride level of the normal individual remains unchanged, whereas in the postoperative patient the level goes up because the normal patient puts out 2.3 liters of the 3 liters given, while the postoperative patient puts out only 1.2, due to oliguria.

If glucose and water are given, the opposite occurs. The level of plasma sodium in the normal patient remains normal, whereas in the operated patient the sodium level drops, also due to the fact that the water given is retained.

Figure 2 combines data from a number of papers by Drs. F. A. Coller and J. D. Stewart,

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Presented at the thirty-sixth (St. Louis) annual Assembly of the Interstate Postgraduate Medical Association.

and shows normal range of plasma chloride during the first 24 to 96 postoperative hours. If you give hypotonic sodium chloride or glucose and water, the level falls; with isotonic sodium chloride, it rises.

In 1938 Dr. F. C. Helwig showed the danger of water intoxication when a patient received large amounts of water. All clinical signs, including coma, were corrected by giving hypertonic salt solution intravenously. But in 1951 another paper reported a similar though less pronounced overload of water in patients after operation, and convulsions and coma followed the administration of several liters of glucose and water daily (Figure 3).

Our own data are given in Figure 4 and show the difference in the level of plasma sodium in the series of cases given glucose in water and those given glucose in saline; with the latter it goes up, with glucose and water it goes down.

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As a result of these observations, it is clear, I think, that the postoperative patient cannot handle extra loads of either water or salt, and one may well ask: Why is it necessary to impose the water and salt load upon the postoperative patient? The answer is, of course, that one should not, yet such excessive fluid administration is not uncommon and continues to produce preventable and wholly unnecessary complications.

Before discussing our method of meeting this difficulty, I would like to discuss briefly the problem of potassium. In recent years it has become apparent that postoperative electrolyte disturbances are not limited to sodium chloride, and that the part played by potassium may be decisive. Under normal conditions the adult is in what we call potassium balance; that is, the amount lost equals the amount of his intake. Even in simple starvation, though there is no potassium intake, evidences of potassium deficiency are rare or nonexistent because the amount lost in the urine is merely a byproduct of the breakdown of tissue cells to supply energy.

The postoperative patient loses much larger amounts of potassium in his urine than can be accounted for by the breakdown of tissue cells. This is particularly true during the first 24 hours after operation, regardless of the water and salt intake, although the loss is greater when saline is given than when water and glucose are given. If the patient vomits, additional but not very large amounts of potassium are lost in the fluid, which tends to have a low concentration of potassium. However, saliva contains a higher concentration of potassium, but not as high as diarrheal fluid which tends to have a content up to 50 mEq., or about 2 gm. per liter. Urine, on the other hand, may contain up to 150 mEq. per liter, or 6 gm. of potassium. By our own calculations, the amount of potassium lost in the average surgical case on a potassium-free intake does not exceed 200 mEq., or about 15 gm. as potassium chloride, which is not enough to produce deficiency in the average case, and certainly not in a period of three to four days.

We have learned that manifestations of potassium deficiency do appear when the loss is sufficiently large to lower significantly the plasma and the intracellular level. Unlike sodium, which is almost entirely extracellular, the great-

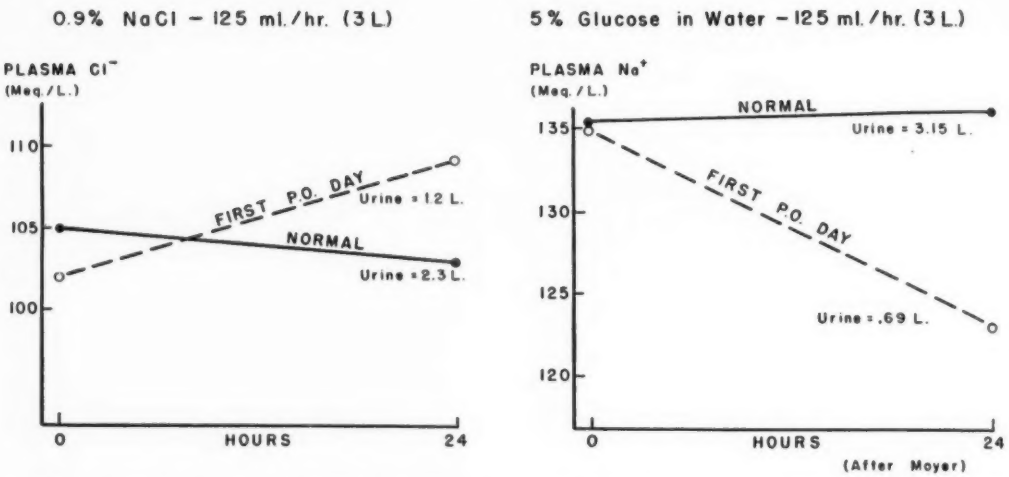

FIGURE 1. Effect on plasma chloride level of 3 liters of saline and 3 liters of glucose, first in a normal person and then in a patient the first day after abdominoperineal resection.

est part of the potassium in the body is contained within the cell. Thus the plasma potassium level is quite low, which means that in the entire extracellular space there is only about 2 gm. of potassium ion. However, a fall in the plasma level is probably responsible for many changes in the electrocardiogram, whereas most other manifestations—weakness and so on—are due to intracellular deficits which may occur even when the plasma level is normal. In most cases, however, potassium is deficient in both extracellular and intracellular spaces.

The total amount of potassium loss capable of producing manifestations is inadequately known, but it is probable that at least 300 mEq., that is 12 gm., of potassium ion (24 gm. of potassium chloride) must be lost before the patient has such obvious clinical signs as prostration, orthostatic hypotension, muscular weakness, abdominal distention, gastrointestinal atony, and so on.

In summary, one might say that, in the uncomplicated case, postoperative electrolyte disturbances may be caused by the injection of excessive amounts of either water or salt, as well as by failure to administer potassium. In other words, postoperative patients differ in two ways: They lose potassium; they cannot handle extra loads of water or salt. In order to avoid such disturbances, the author and his co-workers have developed a formula for what we call a basic minimum water and electrolyte intake for the average adult postoperative patient unable to take fluids by mouth following an uncomplicated surgical procedure.

This minimum basic intake consists of 2 liters of water per day, which under standard conditions is sufficient to provide for insensible losses, for adequate urinary excretion, and even for the replacement of a small amount of vomitus or diarrhea. In these 2 liters are dissolved the electrolytes (salt mixture) as shown in Table 1. To the 2 liters of water containing this salt mixture we add 100 gm. each of glucose and amino acids plus a mixture of vitamins including 1 gm. of vitamin C. Additional fluid is added only when it is needed to replace abnormal losses through the gastrointestinal tract, the skin or the lungs. This routine is changed only when the patient develops oliguria or anuria. In such a case the intake is limited entirely to the insensible loss, which normally represents about 1 liter of water, to which is added 100 gm. of glucose.

The results when the patients were given 2 liters of water pre- and postoperatively are given in Figure 5; each dot represents one patient—one 24 hour collection of urine. They all had a

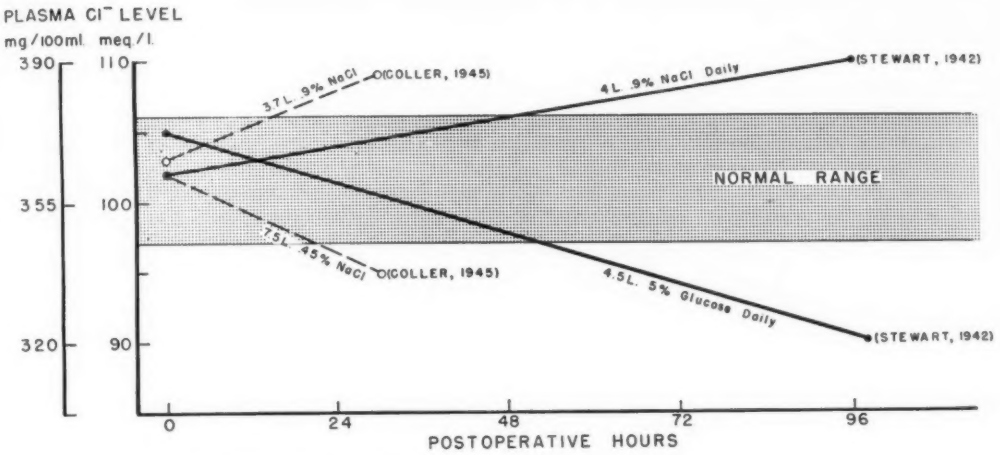

FIGURE 2. Postoperative plasma chloride levels after various water and salt intakes.

very adequate urinary output except on the first postoperative day, which is almost normal because it is really a physiologic phenomenon. These patients were not edematous; some of them were a little dry, but I always prefer to keep the patients a little dry rather than too wet.

Our formula is made up in ampules, 40 cc., and if you figure it out it furnishes about 30 mEq. each of sodium, potassium, phosphorus and chloride (Table 1).

The balance data obtained by using this salt mixture were published in detail elsewhere (*Annals of Surgery*). Briefly, in a series of 10 or 12 patients, some of them lost a little sodium, some of them retained a little sodium, but in general the average balance was about zero. On the first postoperative day they were in negative potassium balance due to the extensive loss of potassium on the first and second days, but even at that the loss was not sufficient to cause symptoms, and in general we can say that their potassium needs were met.

Such therapy will tend to prevent water and electrolyte deficits after operation in the average uncomplicated case. However, certain disturbances develop due to excess losses because of vomiting, etc., which have not been corrected before you see the patient or even afterward.

I would like, therefore, to present two cases, one of sodium deficiency and one of potassium deficiency. I mention sodium and potassium because I do not believe that one can tell all one needs to know about electrolyte deficiencies by merely measuring the chloride level. Much of our new knowledge has been obtained by the use of the flame photometer which enables us to measure the plasma potassium and the plasma sodium. However, I will try to interpret the data so that you may be able to treat patients with sodium and potassium deficits even though you do not have a flame photometer. I do not believe one needs an elaborate set of instruments to practice surgery. I think part of our job—those who are equipped with these instruments—is to translate the data we obtain to bedside manifestations and simple clinical measurements that can be used by everybody.

These two cases illustrate (1) a case of sodium deficit requiring hypertonic sodium chloride and (2) a case of potassium deficit. I might say that since we have been treating patients to prevent sodium and potassium deficits, we seldom see such patients any more. Formerly in a period of six months at the Barnes Hospital we found 30 or 40 cases of potassium deficiency; now we have a hard job finding a case to demonstrate to visitors, because potassium deficiency is now prevented rather than corrected.

The first patient had developed a postoperative intestinal obstruction. When I saw him he

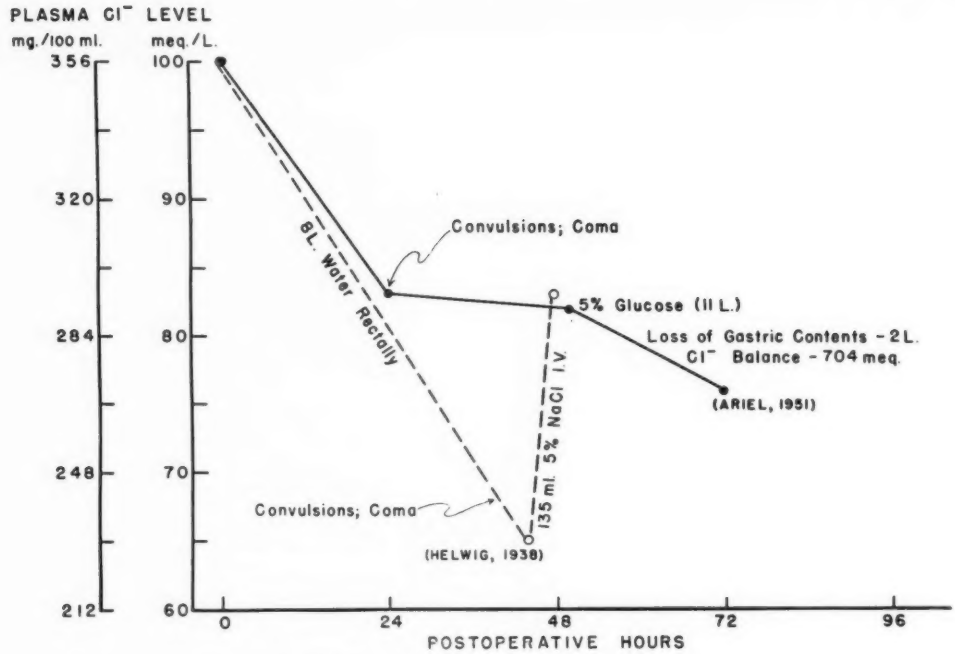

FIGURE 3. Postoperative plasma chloride levels following intake of water without salt.

was prostrated with hypotension; his blood pressure responded to transfusion but he was mentally obtunded, his abdomen was distended and there was roentgenologic evidence of intestinal obstruction. Because of his prostration I felt that he had a strangulation. His plasma chloride level was low; the potassium level was normal. He was not putting out any urine; his nonprotein nitrogen level was high; he had uremia, really, and I was afraid to operate on him, but we measured his plasma sodium level and found it to be down to 118 mEq. per liter instead of the normal of about 145. We learned that he had been drinking water and, between episodes of vomiting, water alone had been given as glucose in water instead of saline, and therefore he had water intoxication.

Since I decided to operate on him because of the danger of strangulation, we gave him 400 cc. of 5 per cent sodium chloride during the next few hours. Immediately the sodium level rose almost to normal. He improved mentally but he did not put out any urine; I still felt we had to operate on him, which we did. I am sure if we had not given him the hypertonic sodium chloride he would have died and the cause of death would have been "intestinal intoxication." Actually he did not have a strangulation but simply a mechanical obstruction which was relieved. After operation his nonprotein nitrogen began to fall with the onset of a brisk diuresis, which amounted to nearly 3 liters despite no further administration of fluid. The only thing this man complained of in his postoperative period was the fact that he could not sleep because he had to pass so much urine.

This, therefore, represents a case of pure sodium deficiency, water intoxication and prerenal azotemia, rapidly corrected by giving the patient enough sodium chloride, which he did not have, without too much water, of which he had an excess. He could not excrete this excess water (he was actually edematous) because the kidney cannot excrete urine without sodium and it was retaining this sodium to such an extent that it would not even excrete water. Indeed we noticed that the sodium content of the urine was very low.

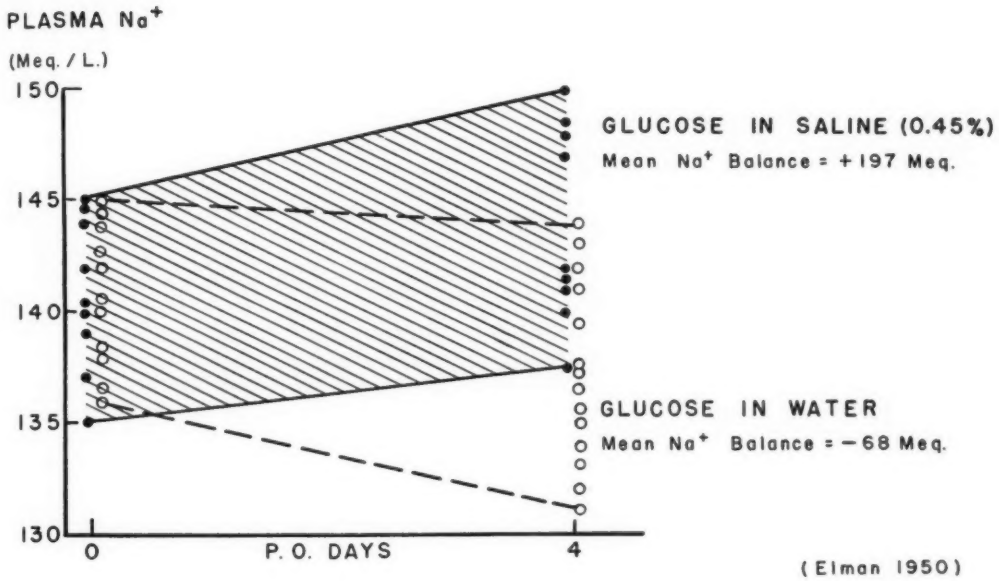

FIGURE 4. Postoperative plasma sodium levels after daily intake of glucose in water and of glucose in saline.

THE next case is also dramatic and illustrates a failure of routine care. The patient had a perforation of a diverticulum. She had a very low chloride level and she also had a fairly high carbon dioxide level; in other words, she had so-called hypochloremic alkalosis. She was weak and prostrated. We gave her lots of sodium chloride. She did not improve very much. We thought perhaps she ought to be operated on; she was. We found a large abscess, which was drained, and we did a colostomy. The colostomy was done very well technically but didn't function. She continued to lose plasma chloride and her carbon dioxide continued to go up. We could not understand why the colostomy didn't work and the patient didn't respond. On the twenty-seventh day she developed tetany, which responded very well to intravenous calcium, but she was still just as sick as she was before.

Then (this was in the early days) we got a potassium level, which was down to 1.2 mEq.—the normal is 5. We began to realize, then, that this patient was suffering from potassium deficiency. It is very simple to give potassium, but it takes many days to correct potassium deficits, largely because you cannot give very much and the kidney excretes much of what you give for a number of days.

In this case, after about 10 days the potassium level came up, the chloride level was restored to normal, the carbon dioxide came down to normal, the colostomy began to function, and the patient became transformed from a weak, apathetic, stupid patient to a really bright, alert individual who was eager to get well.

I would like to discuss briefly some questions concerning dosage. The actual amount of sodium chloride needed in patients who have been vomiting, in the absence of potassium deficits, may be roughly calculated from knowledge of the plasma chloride level. However, if one employs the volume of the extracellular space in this calculation, the amount usually will be insufficient because, as pointed out by Peters, one should use the total body water rather than the extracellular volume in calculating the amount of salt needed to correct a deficiency.

To take a simple example, if a patient has a plasma chloride of 80 mEq. per liter, which is 20 under normal, the average extracellular space would be about 13 liters, which, times 20, would be 260 mEq., or 2 liters of isotonic

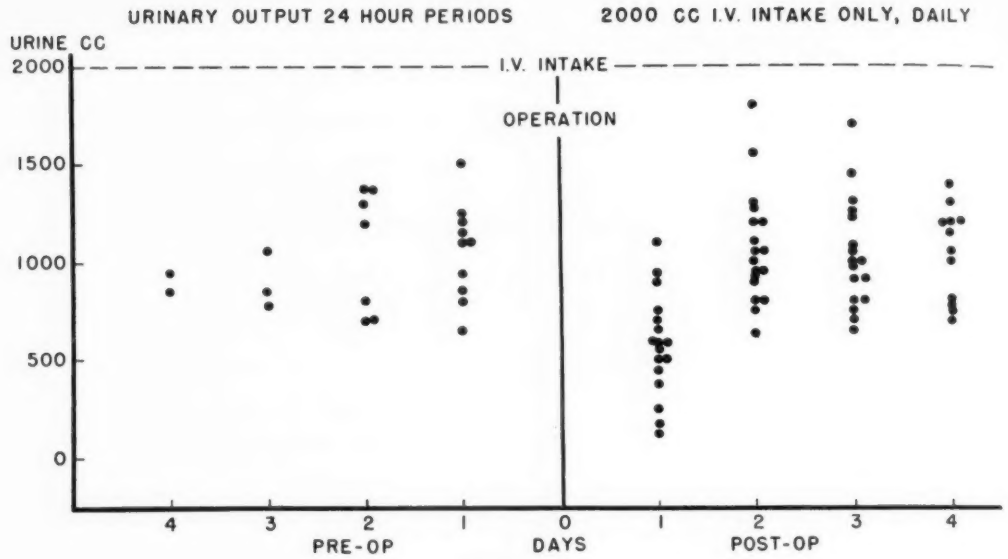

FIGURE 5.

saline, which is certainly insufficient. By using the normal total body water in such a case, the amount would be 50 liters, times 20, or 1000 mEq. of sodium chloride. In isotonic solution, this would mean 7 liters, which probably would correct the deficiency provided the patient were extremely dehydrated and exhibited no evidence of water excess. On the other hand, if the plasma sodium level was also low, indicating a relative water excess, the same amount of sodium chloride would be given in concentrated form, and this would mean 1.2 liters of 5 per cent sodium chloride. In actual practice such an amount has proved too much. For example, in the case I just described, 400 cc. of 5 per cent sodium chloride restored the plasma chloride level to normal, thus indicating that in this case over a liter would have been excessive. Although we do calculate the sodium chloride need on the basis of the total body water, in actual practice we tend to give much less and then observe the clinical and chemical response to therapy before giving additional amounts.

I would like to emphasize the need for observing the patient carefully and giving him additional amounts if his clinical condition seems to demand it.

As to potassium, the problem is more difficult because this element is almost entirely intracellular and we have no direct way of measuring the intracellular fluid. Measurement of the plasma level is useful, and so is the electrocardiogram. However, there are many exceptions in studying electrocardiograms. Potassium in general is given until the plasma level is restored to normal or the changes in the electrocardiogram have disappeared. However, this does not necessarily mean that the intracellular deficits are corrected. For this purpose, the achievement of potassium balance is important. Potassium is given until the urinary output equals the intake.

Unfortunately, large amounts of potassium cannot be given intravenously because any concentration of potassium chloride above 4 gm. per liter produces painful phlebitis. Thus 8 to 12 gm. of potassium chloride represents in general the maximum daily dose for patients being given 2 to 3 liters of water a day. It is known that many patients require as much as 25 to 50 gm. of potassium chloride. Usually twice this amount must be given because much of the salt administered is not retained but is lost in the urine despite a deficiency. For these reasons, it usually requires much potassium chloride and a week or more to correct intracellular potassium

deficits. This is probably just as well because potassium therapy may be dangerous if the solution is given too rapidly, that is, at a rate of more than 4 gm. per hour, or if the patient is suffering from oliguria and particularly anuria. The reason for this is that the plasma level of potassium must not be increased too rapidly lest cardiac stoppage occur. Frequent electrocardiographic readings are also an effective guard against a too dangerous elevation in the plasma level.

In our own experience with the administration of potassium to many patients we have never seen any evidence of cardiac irregularities. I know of only one case of cardiac stoppage, and that occurred when someone by mistake rapidly injected 4 gm. of potassium chloride in concentrated solution.

TABLE 1

SALT MIXTURE

	Grams per cubic centimeter
Sodium chloride	0.05
Dibasic potassium phosphate	0.062
Monobasic potassium phosphate	0.014

Daily dose*: 40 cc. added to 1 liter of intravenous solution.

*This furnishes the following:

Na = 34 mEq.	P = 0.569 gm.
K = 33 mEq.	Cl = 34 mEq.

THOSE of you who do not have flame photometers and other biochemical equipment may still employ fairly adequate therapy. The best guides in such a situation will be, first, knowledge of the factors which resulted in the deficiency, and second, the clinical response of the patient to therapy.

First, a good history of the patient's intake and output will often furnish a clue as to the type of solution required for the correction of water and salt depletion. For example, a patient who has merely vomited and who has been unable to retain any fluid at all would undoubtedly require an early replacement of the vomitus, which would be done readily by the injection of isotonic sodium chloride solution. If the difficulty has been present for several days, potassium chloride should be added to the fluids. If the patient has been unable to take any fluid and has had no vomiting or diarrhea, hypotonic sodium chloride should be given, that is, one containing about 2 to 4 gm. of sodium chloride and about 2 gm. of potassium chloride per liter. In this case, the salt mixture previously mentioned added to 5 per cent glucose and amino acids would be most appropriate.

If the patient has vomited and then ingested pure water or if the dehydration has been mistakenly treated by glucose without electrolytes, hypertonic (5 per cent) salt solution should be given. Patients who have not vomited at all and have been given large quantities of water without salt would fall in the same category.

Second, the clinical response to therapy is also a simple guide. A rapid effect is usually seen in patients with water intoxication and those who require hypertonic salt. Here the injection of, say, 200 cc. of 5 per cent sodium chloride may produce striking improvement in the mental apathy of the patient and be followed by a prompt diuresis. If the reaction is inadequate, an additional injection of 200 cc. of 5 per cent sodium chloride may be given; this will be followed by further clinical improvement. It should be emphasized that salt should not be given and may prove harmful to patients with anuria due to renal disease just because they show hypochloremia. A low plasma chloride in such patients often results from causes other than loss of fluid by vomiting.

In the case of potassium deficiency, improvement in the sensorium, disappearance of muscular weakness, relief of distention, cessation of nausea and vomiting, and the return of normal gastrointestinal function, all mean correction of the deficiency, but usually require about 6 to 10 days of therapy before they are completely effective.

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STATISTICAL STUDY

A Review of 527 Mid Forceps Deliveries

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Mid forceps were used in 4.48 per cent of all deliveries at The New York Polyclinic Medical School and Hospital during the 10 year period from 1941 through 1950. There were no maternal deaths, the gross fetal loss was 3.03 per cent (corrected to 2.27 per cent), and the maternal morbidity rate was 2.28 per cent. Included in this review are discussions on the types of forceps used, the complications encountered and the causes of fetal and infant mortality and maternal morbidity.

During the 10 year period from 1941 through 1950, 11,777 viable babies were delivered at The New York Polyclinic Medical School and Hospital. Of these, 527 (4.48 per cent) were delivered by means of mid forceps, including a set of twins. Four hundred and sixty of the mothers were private patients and 66 were ward patients, with 61 different physicians listed for the group. Three hundred and fifty-six women were primiparas and 170 were multiparas; the average age for the primiparas was 27.8 years, for the multiparas 31.9 years. Thirty-eight women were classified as elderly primiparas. Thirty-three patients were delivered under caudal anesthesia; general inhalation anesthesia was used for the remainder. The average birth weight for the infants was 3,611 gm. (7 pounds, 8 ounces), the smallest weighing 2,060 gm. (4 pounds, 6 ounces) and the largest weighing 5,210 gm. (11 pounds, 7 ounces). Fourteen patients were delivered after a period of gestation of 37 weeks or less.

This paper is based on a talk presented before the Section on Obstetrics and Gynecology, New York Academy of Medicine.

At this institution forceps deliveries are divided into five categories depending on the station of the fetal head in the pelvis: (1) low forceps, (2) low-mid forceps, (3) mid forceps, (4) high forceps and (5) forceps to the unengaged head (Table 1). A critique of this system of classification soon will be presented by one of us (E.H.D.). A mid forceps delivery is defined as one in which the forceps are applied to the head in the mid station, i.e., with the leading bony point at or just above the plane of the ischial spines, the hollow of the sacrum nearly filled by the fetal head, and the biparietal diameter below the inlet. In determining the station, account is taken of moulding, asynclitism, extension of the head and deformities of the pelvis as possible misleading factors.

Any delivery in which the fetal head did not meet these requirements was classified as a high forceps delivery* and is not included in this analysis. Deliveries of vertices with the leading bony point past the ischial spines and the fetal head filling the hollow of the sacrum

*No forceps were used when the head was unengaged.

TABLE 1

VIABLE BABIES DELIVERED BY MEANS OF FORCEPS AT NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL (1941-1950)

	NUMBER	PERCENTAGE OF DELIVERIES FOR 10 YEAR PERIOD (11,777 TOTAL)
Forceps to unengaged head..	0	
High forceps.....	11	0.09
Mid forceps.....	527	4.48
Low-mid forceps	2,391	20.3
Low forceps.....	3,767	31.98
TOTAL	6,696	56.85

but not yet resting firmly on the perineum between contractions were classified as low-mid forceps. Low forceps deliveries were those in which the leading bony point was resting on the perineum between contractions and the sagittal suture was in or near the anteroposterior diameter.

Over the 10 year period, the yearly incidence of mid forceps deliveries has varied but little, the highest being 6.8 per cent in 1943 and the lowest being 2.88 per cent in 1949 (Table 2).

TABLE 2

MID FORCEPS DELIVERIES AT NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL—PERCENTAGE OF TOTAL NUMBER OF DELIVERIES PER YEAR (1941-1950)

	PER CENT OF TOTAL DELIVERIES
1941	5.53
1942	4.31
1943	6.80
1944	5.03
1945	4.01
1946	5.97
1947	4.61
1948	3.23
1949	2.88
1950	3.74

Indications for use of mid forceps were as follows: *fetal distress,* 96 cases, including 3 premature separations of the placenta, 3 instances of prolapse of the cord and 1 marginal placenta previa; *malposition,* 149 cases (80 listed as transverse arrest, 52 as persistent right occipitoposterior or left occipitoposterior, 7 as extended heads, 5 as persistent direct occipitoposterior, 3 as face presentations with posterior chin and 2 as brow presentations); *ineffectual labor,* 148 cases, listed variously as prolonged second stage,

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prolonged labor, unsatisfactory progress, desultory labor, cervical dystocia and primary and secondary uterine inertia; *maternal distress,* 26 cases, including 7 patients with cardiac disease, 5 with severe pre-eclampsia and 1 with arrested pulmonary tuberculosis; *elective,* 108 cases.

All the patients in the 108 elective cases were on the private service. In these, indications for mid forceps delivery were not sufficiently well defined to be included in any one of the groups of patients with the complications already listed. However, in the opinion of the operator, delivery by mid forceps was considered advisable in anticipation of the possible development of one or more of these complications. It is interesting to note that in these private elective cases there were no fetal or maternal deaths and the one instance of maternal morbidity was due to suppurative mastitis.

The presentations at time of delivery were as follows: direct occipito-anterior 7, left occipito-anterior and right occipito-anterior 34,

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left occipitotransverse and right occipitotransverse 259, left occipitoposterior and right occipitoposterior 204, direct occipitoposterior 18, posterior face 3, and brow 2.

In 445 deliveries, only one instrument was employed; in the remaining 82 cases, two instruments were used—the first for rotation, the second for traction (Table 3). The Tarnier forceps have not been used in any case for the past five years.

The gross fetal and infant mortality in these 527 mid forceps deliveries was 16 (3.03 per cent). This figure compares favorably with a gross mortality rate of 3.03 per cent for the 3,428 normal spontaneous deliveries during the same period of time, but it compares unfavorably with the mortality rate of 1.09 per cent in the total 6,696 forceps deliveries (Table 4). Excluding 4 infants with congenital abnormalities which were incompatible with life, the corrected fetal and infant mortality for mid forceps delivery was 12 (2.27 per cent).

There were no maternal deaths in this group. The maternal morbidity rate of 2.28 per cent (12 patients) compares unfavorably with the morbidity rates for both the normal spontaneous deliveries and the total forceps deliveries, which were 1.34 per cent and 1.03 per cent respectively. The causes of fetal and infant mortality and maternal morbidity are shown in Tables 5 and 6.

TABLE 3

TYPES OF FORCEPS USED IN 527 MID FORCEPS DELIVERIES AT NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL (1941-1950)

TYPE	ALONE	IN COMBINATION
Kielland's	345	73
DeWees	35	52
Hawks-Dennen	25	17
Barton	18	9
Tarnier's	12	6
Elliot	5	4
Tucker-McLean	4	2
Simpson's	1	1

COMPLICATIONS listed for this group of patients were: premature rupture of the membranes 53, partial premature separation of the placenta 4, leiomyomata uteri 7, pre-eclampsia 8, contraction ring 1, cardiac disease 7, congenital

TABLE 4

COMPARISON OF MORTALITY AND MORBIDITY RATES FOR FORCEPS DELIVERIES AND FOR SPONTANEOUS DELIVERIES (1941-1950)

METHOD OF DELIVERY	FETAL AND INFANT MORTALITY				MATERNAL MORTALITY		MATERNAL MORBIDITY	
	Gross		Corrected*		*Number*	*Per Cent*	*Number*	*Per Cent*
	Number	*Per Cent*	*Number*	*Per Cent*				
High forceps (11)	0				0		0	
Mid forceps (527)	16	3.03	12	2.27	0		12	2.28
All forceps (6,696)	73	1.09	46	0.68	6	.089	69	1.03
Spontaneous (3,428)	104	3.03	44	1.28	1	.029	46	1.34

*Excluding monsters and macerated babies.

TABLE 5

CAUSES OF INFANT MORTALITY IN 527 MID FORCEPS DELIVERIES (1941-1950)

Stillbirths	9
Microcephalia with meningocele	1
Corrected number of stillbirths	8
Intra-uterine asphyxia	6*
Tentorial tear	1
Intracranial hemorrhage	1
Neonatal deaths	7
Dextrocardia with diaphragmatic hernia	1
Hydrocephalia with meningocele	1
Rudimentary Siamese twin	1
Corrected number of neonatal deaths	4
Pneumonia	1
Atelectasis	1
Cause undetermined	2
TOTAL	16 (3.03 per cent)

*Includes 1 separated placenta, 1 prolapsed cord.

Autopsy was performed on 12 of the 16 infants (75 per cent)—7 of the stillbirths and 5 of the infants who died neonatally.

TABLE 6

CAUSES OF MATERNAL MORBIDITY IN 527 MID FORCEPS DELIVERIES (1941-1950)

CAUSE	NUMBER OF PATIENTS
Mastitis	5
Undetermined	3
Endometritis	2
Pyelonephritis	1
Upper respiratory infection	1
TOTAL	12 (2.28 per cent)

syphilis 2, retained placenta 4, marginal placenta previa 1, chronic polyhydramnios 1, postpartum hemorrhage 5 (mild 3, moderate 1, severe 1). There were 18 sulcus tears. Twelve patients required repair of lacerated cervices. One patient had a third degree laceration of the perineum. There were no instances of injury to the uterus or bladder.

We believe that there is and will continue to be an important place for mid forceps deliveries in modern obstetrics. However, the success of the operation depends on the judgment and skill of the operator, proper anesthesia, adequate assistance and the correct choice of instruments. The incidence of the use of mid forceps in those patients with unsatisfactory labor may be somewhat lowered in the future because of the increased use of oxytocics in cases of uterine inertia. X-ray pelvimetry, especially a side view of the pelvis, gives invaluable aid in establishing the station of the biparietal diameter as well as in demonstrating disproportion.

The use of mid forceps has fallen into disrepute in some sectors largely because of errors in judging the exact station of the fetal head. Consequently, unsuccessful and traumatic high forceps deliveries often are improperly labeled mid forceps. In addition, errors in decision are made in attempting forceps deliveries in complicated cases in which it would be better to perform cesarean sections.

CONCLUSIONS

The success of mid forceps deliveries depends to a large extent on the judgment, experience and ability of the operator. The type of pelvis, size and attitude of the fetal head and the condition of the cervix should meet the requirements necessary for a successful delivery by mid forceps. If these factors do not favor use of mid forceps, best results will be obtained by cesarean section. If vaginal delivery is decided upon, the choice of instrument will depend on the type of pelvis, amount of moulding and the position and attitude of the fetal head.

Determination of the exact station of the fetal head is necessary to exclude high forceps and also low-mid forceps. In the complicated case, a lateral roentgenogram of the pelvis aids in this determination. With proper diagnosis of station the hazards of the usually undesirable high forceps operation may be avoided. At the same time the less difficult low-mid forceps operation is not included and the results will not show a falsely favorable impression of the risk of the true mid forceps delivery.

Mid forceps deliveries can be extremely dangerous, especially when the case is poorly selected and the operation poorly executed. However, this is not sufficient reason to discard a valuable procedure which, when certain conditions are met, can produce satisfactory results.

GENERAL PRACTICE *Review*

To provide the general practitioner, as well as the specialist with concise, readily available information on the latest, proved methods of treatment of conditions commonly encountered in an average practice, POSTGRADUATE MEDICINE *offers this department to its readers. It is not, of course, intended to present these discussions as the only acceptable therapeutic procedures to be used, but rather to offer simple regimens and recommendations based on the extensive experience of the physicians who prepared these summaries.*

Acute Otitis Media

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ACUTE infections of the middle ear continue to be a serious problem in the everyday practice of medicine. Although the widespread use of the various potent antimicrobial agents has greatly modified these inflammatory processes, the incidence of complicating chronic otitis media and middle ear fibrosis has not been decreased correspondingly. It seems probable that, when statistics become available, they will show not only a relative increase in the chronic suppurative processes, but more important, an increase in resultant progressive deafness. The reason for this becomes evident when the average present day treatment of acute middle ear infection with antibiotics alone is scrutinized.

Antimicrobial therapy, which produces a rapid drop in fever and relief of pain together with the marked decrease in immediate serious complications, has resulted in the physician's being lulled into a false sense of security. This complete faith in antimicrobial therapy has led to the neglect of other useful therapeutic measures and has also produced a tendency to carry poorly responding cases into a danger period. Valuable time is lost either by shifting to other antibiotics or by increasing doses, when a well performed myringotomy or simple mastoidectomy would have resulted in prompt clearing of the infection with preservation of hearing.

In evaluating present day treatment of acute middle ear disease, it is important to consider that approximately 80 per cent of the acute infections will subside quickly with the simple treatment of bed rest, heat to the ear, and vasoconstrictors. In this group it is obvious that sulfonamides or antibiotics are unnecessary and useless. However, the remaining 20 per cent provide the serious problem in therapy. It is often difficult to determine the severity of the infection at its onset, and many of the important early hours of effective treatment will be missed if one waits for time to determine the seriousness of the infection. From this it is evident that each case must be evaluated individually and treatment individually instituted.

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ALTERED PHYSIOLOGY PREDISPOSING TO ACUTE OTITIS MEDIA

Before rationally considering any plan of treatment, there must be an understanding of the altered physiology that predisposes to infections in this area. In the vast majority of middle ear infections, the preliminary infection begins in the nose and nasopharynx. Direct extension involving the ostia, plus the general bathing of the area with infected secretion, result in an acute eustachian salpingitis. The infection then spreads along or through the tube to reach the middle ear. There the severity of the infection is determined by the virulence of the organism, the resistance of the host, and local anatomic factors. Thus in infancy and early childhood, the shorter, relatively wider and more horizontally placed eustachian tubes result in an increased incidence of middle ear infection.

Adenoidal infection, due to its close proximity to the eustachian tubal orifices, plays a major etiologic role in middle ear infections. This is openly apparent in the child with complete or almost complete postnasal blockage, but is not so apparent in the individual with small masses of infected lymphoid tissue lying in Rosenmüller's fossae. In these the recurrent bouts of acute otitis media are repeated extensions of the continued infectious process present at the nasopharyngeal ostium. These infected adenoid masses are not limited to childhood but often persist through adult life, producing recurrent infections and gradually destroying the middle ear structures.

Infected material from the nose and paranasal sinuses plays an important role in producing chronic infection in the nasopharynx with resultant hypertrophy of the adenoidal elements. In childhood this is more common than is generally realized. E. Prince Fowler, Sr.,[1] in 1929, in a study of hearing deficiencies in children, noted that 87 per cent showed roentgenologic evidence of sinusitis. Although there are no recent similar studies available, clinical experience indicates that a similar high percentage is present today in children having recurrent attacks of acute otitis media.

The predisposition of the allergic patient to paranasal sinus infection, together with the direct allergic involvement of the mucosa of the eustachian tube itself, are definite factors in the production of otitis media. Day[2] noted this in a study of the etiology of chronic eustachian obstruction and reported that many resistant cases responded to adequate allergic management.

Heredity seems to influence a predisposition to recurrent attacks of otitis media. In a great part they are the result of familial tendencies to lymphoid hypertrophy with the secondary involvement of the eustachian tube. In these cases, extra precautions with respiratory infections plus careful surgical removal of the lymphoid tissue in the areas of Rosenmüller's fossae will offer the greatest protection.

PATHOLOGY OF ACUTE OTITIS MEDIA

In an acute otitis media, the pathologic picture is rapidly progressive. At the onset there is edema of the submucosa with an increase in the capillaries and small arterioles. Polymorphonuclear leukocytes and plasma cells collect in the tissue spaces. At this stage, the drum membrane is usually slightly retracted and shows a reddening, especially about its periphery. The patient complains of a sense of fullness and a dull, aching pain of moderate severity. This phase, which is usually very transient, is followed by an increase of cellular infiltration, and fluid containing blood and polymorphonuclear cells appears in the middle ear cavity. As this exudate increases, the pain becomes more severe. The drum membrane soon becomes intensely reddened with obliteration of normal landmarks. The collection of exudate and swollen mucosa produces bulging of the drum surface outward. The extent of this is largely dependent upon the type and virulence of the bacteria present. In a rapid, severe infection, a perforation soon appears at the site of maximum bulging. If this does not appear quickly, pressure necrosis may destroy a large segment of the drum, often leaving a large, permanent perforation.

In a fulminating infection this entire process may be very rapid with only a few hours elapsing between onset and rupture, while in a lesser infection 24 to 36 hours may pass before perforation takes place. In the more severe infections the process spreads outward into the ad-

joining mastoid cells and there produces the same pathologic changes with collection of exudate. In this area, the combination of soft-tissue swelling and the rapidly forming exudate is sufficient to fill the cavities and block the blood supply. If this process continues, it produces a breaking-down of the bone trabeculae between the cells and marks the beginning of an irreversible phase.

At this time, antimicrobial therapy alone is of limited value because of the pathologic lack of blood supply to the involved area. Adequate surgical drainage is essential at this stage. A well performed, early myringotomy will often relieve the middle ear and its adjoining mastoid cells of the pressure of the trapped exudate and allow the blood supply to be re-established. This will allow the antimicrobial agent again to exert its full influence. If the drainage remains inadequate and the disease process remains unchecked, the pathologic changes advance to a stage where surgical removal of the necrosing tissues is necessary if one is to prevent chronic infection and to salvage hearing.

In this group, antimicrobial therapy alone will lower the immediate danger of central complications, but it will also produce a misleading masking process. The reduction of fever and easing of symptoms cover an underlying low grade necrosis which will result in a chronic otitis media and mastoiditis and will eventually destroy hearing. In these, the combination of a well performed, simple mastoidectomy together with the indicated biotherapy would have resulted in rapid clearing and, usually, complete healing in five to seven days, leaving an intact, normally functioning ear. The endaural approach as advocated by Lempert is preferred, as it offers direct accessibility to the diseased area with a minimum of scarring.

BACTERIOLOGY

In considering the use of antimicrobial therapy for acute infections of the middle ear, it is imperative that a full study of the various etiologic organisms, their nature and sensitivities, be made. It is difficult to evaluate statistically the results of this type of therapy, as today almost every acute case will have received one or several antibiotics and often sulfonamide therapy before cultures are taken. Grebelius and Sjoberg in 1949[3] reported on 336 consecutive cases of acute otitis media in Sweden and found only 2.1 per cent of these cases cultured organisms insensitive to penicillin. The resistant strains were largely hemolytic staphylococci (coagulase-positive) which were aureomycin-sensitive.

Today in the United States it is doubtful that this high percentage of penicillin-sensitive organisms exists. However, during the past three years we found somewhat representative figures in cultures taken on patients with acute otitis media. Cultures were obtained from the myringotomy knife if paracentesis was performed or from the neighborhood of the perforation if the ear was discharging. In others with an acute ear without bulging, cultures were obtained from the nose, posterior choanae, or nasopharynx. One hundred consecutive cases were studied and it was found that 76 per cent of the organisms cultured showed definite penicillin sensitivity, 13 per cent showed definite aureomycin sensitivity (most of this group were hemolytic staphylococci which were insensitive to penicillin), 8 per cent showed varied sensitivities, with terramycin, CHLOROMYCETIN® and streptomycin playing major roles. Two cases (2 per cent) showed no inhibition by any of the available antibiotic or sulfonamide preparations. While this small series of cases is of little statistical significance, clinical results seemed to follow the outlined pattern closely. The great majority of infections cleared rapidly when given proper management of the ear plus adequate doses of the indicated antibiotic.

PLAN OF EXAMINATION

In adopting a plan of therapy in acute otitis media, it is essential that each case be individualized and treatment prescribed accordingly. Although many authorities consider it undesirable to start antimicrobial therapy before any results of bacteriologic cultures are available, the effectiveness of early penicillin therapy in a great majority of severe, acute infections of the middle ear is undeniable. These facts were seriously considered in formulating the following therapeutic plan.

After eliciting a history of the onset and char-

acter of the present infection, a careful examination of the auricle, external auditory canal, tympanic membrane and middle ear is carried out. Good illumination and, if necessary, magnification should be used. The mastoid area should be examined for tenderness or swelling. The auricle is manipulated for tenderness especially in the region of the tragus. After cleansing the external auditory canal by carefully wiping away secretion and debris with sterile cotton, the tympanic membrane is inspected. If it appears dull, the surface is carefully wiped clean as often a fine, scumlike layer will obscure an acutely reddened membrane. The degree of involvement of the tympanic membrane itself is determined together with the status of the middle ear. If bulging of the drum membrane is present, the degree and location of the site of maximum bulging are ascertained. If exudate is present, material is obtained for culture before applying alcohol or other antiseptic solutions. After the condition of the ear has been fully evaluated, the status of the nasopharynx, oropharynx, nose, and paranasal sinuses is determined. If possible, a culture is obtained from the area involved.

The general status of the patient's health is studied and other possible coexisting or complicating disease processes are evaluated. The temperature, pulse and other systemic reactions are noted as these are usually an index to the severity of the infection. A blood count and urinalysis should be done, especially in the more severe infection. During examination a more detailed history is obtained. The patient or patient's family is questioned as to the number and severity of similar previous attacks, susceptibility to respiratory infections, known constitutional disturbances and known drug sensitivities.

PLAN OF THERAPY

After evaluating these data, the specific plan of therapy is determined. Bed rest and therapy directed toward the predisposing conditions are of great importance. Active treatment must be directed to the middle ear itself without delay.

In the acutely inflamed ear without bulging, the patient should be put to bed with the usual care of an acute infectious process. Analgesics and sedatives are prescribed as needed to insure rest. Codeine or DEMEROL® is usually indicated. Heat in the form of an electric pad or hot water bottle is of help in relieving pain. Use of ear drops such as AURALGAN® will diminish pain but they are of no value in combating the infection. Attention is given to the nose and paranasal sinuses and treatment prescribed as indicated. Vasoconstrictors are applied as the particular case indicates.

If the patient gives no history of penicillin sensitivity, aqueous fortified procaine penicillin solution (300,000 units procaine penicillin and 100,000 units penicillin G) is given intramuscularly without delay.

If the tympanic membrane shows bulging, a careful, adequate myringotomy is performed and a culture is obtained from the myringotomy knife. Absolute control of the patient is essential in carrying out this procedure. In adults, local anesthesia is usually adequate. The time-proved Bonain solution (equal parts of phenol, menthol and cocaine) has proved satisfactory in my hands with no bad effects. This agent is applied to the surface of the drum using saturated wisps of cotton, taking care that bubbles of air do not prevent the anesthetic from contacting the drum surface. This must be left in place for 15 minutes. General anesthesia may be used if felt desirable and is to be preferred for older children. Any short-acting anesthetic may be employed. Nitrous oxide or VINETHENE® is usually preferred. For infants, wrapping with a sheet in the "mummy" method is all that is necessary.

Preferably the membrana tympani is incised in the posterior half with the incision being made from above, downward. This must be carried out carefully under direct visualization so that underlying structures are not injured.

After myringotomy has been carried out, sterile cotton is placed loosely in the ear to blot up secretions. Instructions are given that this is to be changed whenever the cotton becomes saturated. The patient and family are cautioned about the necessity for sterile technic and warned of the danger of self-induced contamination. Ear drops or irrigations are not used early in the infection. Later the bacteriology and type of secretion may indicate their use. If the secretion becomes thick and viscid, glycerite of hydrogen

peroxide drops may be used twice daily. If the secretion is thin and irritation of the skin of the external canal develops, the canal is carefully wiped clean using a boric acid-alcohol solution. Then a bland ointment (such as BOROFAX®) is gently applied to the skin surface.

An acute perforated tympanic membrane present at the time of first examination is handled on the same plan. A culture is immediately obtained and sterile cotton is placed in the ear. If an otitis externa coexists, the canal wall may be cleansed with an alcoholic solution and a bland ointment applied to the dermal surface. The preliminary bacteriologic sensitivity report will be available, in the great majority, in 24 hours and this will indicate the proper antimicrobial agent to be continued. It is essential that the indicated antimicrobial therapy be given in adequate dosage for a sufficient length of time. A common error is that of discontinuing therapy too early with a resultant recurrence of the infection. A fairly safe criterion is to continue therapy 24 hours beyond what is felt to be the necessary period. In all cases the drum and the amount of suppuration should be carefully watched. If the process does not subside promptly, one should be on guard for a developing bone necrosis.

In all infections not quieting down as expected, roentgenographic studies of the mastoid area should be made without delay. The films should be studied with magnification for evidence of early breakdown of the intracellular bony septa. It is important to watch for sagging of the posterior-superior osseous canal wall because this is pathognomonic of necrosis in the region of the mastoid antrum and is a positive indication for a mastoidectomy. In such cases, prolonged antibiotic therapy may eventually quiet the acute infection, but the tympanic membrane and ossicles are usually irreparably damaged with resultant loss of useful hearing. These poorly responding cases, carried on prolonged antimicrobial therapy, are the source of chronic suppurative otitis media that appears to occur with increasing frequency today. A thorough, simple mastoidectomy performed by a competent surgeon offers a quick and usually complete convalescence with the greatest chance for good hearing.

COMPLICATIONS

Facial paralysis as a result of acute middle ear infection is not common but does occur. The bony covering may be defective in infants and young children and consequently the infection directly affects the nerve. In this younger group without necrosis of the mastoid, adequate attention to the middle ear plus antimicrobial therapy is usually adequate for prompt recovery. In older children and adults, paralysis is the result of bone necrosis with involvement of the bony canal. A mastoidectomy with uncovering of the facial nerve should be performed without delay. The nerve will show prompt recovery of function if this is carried out.

Meningitis as a complication of otitis media follows the same pattern. In the very young the infection may spread through the ununited petrosquamosal suture without necessarily destroying bone, while in the older group it invariably is due to bone necrosis of the dural plate. The meningitis should be brought under control first by proper antimicrobial therapy. This may be sufficient to effect a cure in infants, but in the older group, a prompt, thorough mastoidectomy with removal of the involved dural plate is indicated. In this group it is perilous to depend on antimicrobial therapy alone, as the meningitis is apt to recur again with the increased possibility of a complicating brain abscess. The combination of adequate surgical cleansing of the area plus suitable antimicrobial therapy will result in prompt, complete clearing in a five to seven day period.

Labyrinthitis as a complication of acute otitis media occurs either through direct extension or as a toxic affair. This is best treated by adequate doses of the indicated antibiotic together with the indicated systemic therapy. Intravenous infusion of glucose in distilled water is given until the vomiting is controlled. Surgical intervention is not indicated unless there is reason to suspect an underlying chronic necrosis secondary to previous infections.

Brain abscess is rarely a complication of an adequately treated acute otitis media. In those neglected cases in which it occurs, a thorough mastoidectomy should be carried out with exposure of the dura. Often the abscess can be drained through the involved dura at this time.

The indicated antibiotics should be administered in full doses.

PROPHYLAXIS

After the acute infection has subsided satisfactorily, it is essential that the patient be studied for possible factors that would promote recurrence. Inflammatory lymphoid tissue in the adenoid area, especially that lying in Rosenmüller's fossae, should be surgically removed. Irradiation is of marked value in reducing inaccessible lymphoid tissue about the tubal orifices, but must not be considered as a substitute for adequate surgery. Residual infection in the paranasal sinuses should be adequately treated and allergic phenomena should be brought under control.

Swimming is forbidden until all evidence of infection has cleared and the tympanic membrane has completely healed. Flying is discouraged until eustachian aeration has returned to normal.

The patient should be warned of the danger of reinfection due to improper blowing of the nose. This should be especially emphasized to the patient with an open perforation of the tympanic membrane.

The use of varied vitamin mixtures as a panacea for respiratory infections and recurrent otitis media is useless and wasteful unless a definite deficiency exists. Any dietary defects should be corrected and supplementary vitamin therapy given as indicated. It is essential that the general health of the patient be kept at an optimum level. Often an unnoted secondary anemia will be responsible for the lowered general resistance. When this occurs, adequate doses of iron and vitamin B complex will give spectacular results.

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Anemia in Pregnancy

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The diagnosis and treatment of anemia in pregnancy would seem to be relatively simple. Do a blood count. If anemia is present, give a lot of iron and vitamins to cure it. If there is no anemia, give a little iron and vitamins to prevent it. Is it this simple?

I would like to propose a series of questions concerning the anemias of pregnancy, and then answer them in the light of present knowledge. I believe that in this way it may be possible to change our perspective of this problem from what it has been in the past.

WHEN SHOULD THE GRAVID PATIENT BE CONSIDERED ANEMIC?

Anemia should not be considered to exist in the pregnant patient unless the red cell count falls below 3,500,000 per cubic millimeter, the hemoglobin below 70 per cent or 10 gm. per cubic centimeter, or the hematocrit below 30 volume per cent. These minimum values must be accepted in the light of the known average error in laboratory technics. The hemoglobin estimation is notoriously erroneous, red cell counts less so, and hematocrit determinations most reliable. According to Dieckmann,[1] in estimating the hemoglobin by visual or photoelectric colorimeter, the combined error of pipet and chamber, as well as of the technician, may be 7 to 15 per cent, and the minimum error in the red cell count is over 8 per cent. In hematocrit determinations, using heparin, the error is

approximately 2 per cent.

These lowered values for pregnancy belie an actual increase in the number of erythrocytes and the total amount of hemoglobin. There is an increase during pregnancy of about 23 per cent in the total blood volume, beginning at the first trimester and increasing to near term. The average decrease in blood volume following delivery has been found to be from 1100 to 1800 cc. during the first week. The counts usually are lowest in the third trimester. To allow for individual variations, the blood should be examined in the early stages of pregnancy so that subsequent counts can be compared with the original. It has been our practice to repeat the hemoglobin estimation at the end of the sixth month and again at seven and one-half months.

WHAT TYPES OF ANEMIA OCCUR IN PREGNANCY?

Two main types of anemia may be encountered in pregnancy—the microcytic, which is the common type, and the macrocytic or megaloblastic, which is rare. Other extremely rare anemias are not considered to be within the scope of this discussion.

HOW COMMON IS ANEMIA IN PREGNANCY?

Anemia may be expected to occur in approximately 12 per cent of pregnant patients. Of these, less than 1 per cent will develop the megaloblastic type.

WHEN DOES A PATIENT BECOME ANEMIC?

Anemia may occur at any time during the prenatal course, or it may have been present prior to conception. In patients whose counts were previously normal, however, the highest incidence will occur before the last trimester.

WHAT IS THE CAUSE OF ANEMIA IN PREGNANCY?

Talso and Dieckmann[2] have recently summarized the present knowledge concerning the cause of the anemia of pregnancy:

"During pregnancy an inadequate diet, defective absorption from the gastrointestinal tract and increasing fetal demands have been accused of precipitating this condition. Wolff and Limarzi report a normoblastic hyperplasia of the bone marrow in such cases. These workers and others conclude that these anemias are the result of an iron deficiency and can be correlated by administering simple iron salts. Some workers stress the importance of diet in their prevention and correction. Others indicate that although an adequate diet is important from the standpoint of maternal health, the quality of the diet has little effect on the hematologic findings except in the extremes.

"Changes in the gastrointestinal secretory function occur in most pregnant women, yet no significant correlation exists between the decreased gastric acidity and the incidence of anemia. Further, studies with radioactive materials during pregnancy have revealed that iron is absorbed at 2 to 10 times the normal rate. The amount of iron necessary for fetal growth is negligible during the first 28 weeks and yet it is during this period that there is the greatest fall in the maternal hemoglobin concentration. It is only during the last 12 weeks, when the fetal weight increases from approximately 1000 gm. at 28 weeks to 3400 gm. at term, that the iron requirements of the fetus become appreciable. But even at term, when the actual amount of iron required by the fetus is greater, the total fetal content is only 280 mg., which could be furnished by the iron from 600 cc. of maternal blood. Furthermore, it is during this last 12 weeks that the maternal hemoglobin concentration is either stationary or increased. Thus, during the maximum fetal requirement, the hemoglobin also increases."

In the light of recent experience, the megaloblastic anemia of pregnancy may prove to be caused by folic acid deficiency.[3,4]

SHOULD IRON, VITAMINS AND OTHER DIETARY SUPPLEMENTS BE GIVEN PROPHYLACTICALLY TO ALL GRAVIDAS?

The main purposes of this presentation are (1) to decry again the principle of prenatal care that embodies so-called "routine dosing" of all patients with medicines and dietary supplements during the prenatal period, and (2) to outline a rational, effective and economically

sound method for diagnosis and management of the anemias of pregnancy.

It would seem unnecessary to remind ourselves that, from the standpoint of nutritional completeness, the American diet is probably the best the world has ever known. Why, then, should every pregnant woman's diet contain nutritional supplements merely because such supplements are known and available? American citizens are entitled under the constitution to life, liberty, the pursuit of happiness and, more recently—and commendably so—to the pursuit of health.

In prenatal care, physicians are entrusted with the care of women who are undergoing an age-old physiologic process, one that occasionally may become pathologic. Complete physical examination, adequate laboratory examinations and instruction with regard to hygiene and diet are the nucleus of good prenatal care. Recognition and correction of pathologic states are essential. Prevention of the occurrence of pathologic states is the refinement of modern medicine, but it can be too easily overdone. The cost of medical care has mounted to such a figure that it has been recommended that the health of the American people be made a federal responsibility. Unless we are very cautious and critical of our methods of therapy, such foolishness as supplementing everyone's diet with every known type of nutritional accessory in routine fashion can become such a burden in an already expensive system of medical care that we can price ourselves out of the market by such seeming minutiae.

Great strides have been made in the science of nutrition, and more will be learned in the future. Many obscure diseases have been added to the increasing list of dietary deficiency syndromes. Nevertheless we must continue to be reasonable in the extent to which we attempt to establish a "routine prophylaxis" in prenatal care lest we find ourselves guilty of attempting—at considerable economic loss—to "make a normal process more normal." We believe the most apropos remark that has come to our attention is the quotation taken from the recent article by Youmans[5] in the *Journal of the American Medical Association* with respect to deficiency in vitamins:

"Whenever possible, the cause—ignorance, economic status, alcoholism or other disease—should be removed, but some patients will require maintenance doses of the vitamin or concentrate because of the nature of other illnesses." To this list of causes we would add "lack of interest." Worry, lack of sleep, and social responsibilities in the primipara, and the same together with child-raising and sacrifices in personal demands in the multipara, are real causes of dietary insufficiency. In the economically marginal family, how often do we find that the milk, eggs and fruit go to the children, while mother keeps within the budget by restricting her diet to foods that are less expensive. It is ironic that the patient who is economically unable to have a sufficient diet is ipso facto unable to purchase dietary supplements.

The pregnant woman's diet should be known to her physician. It is very simple for the patient to record her diet as she chooses it naturally for a period of one to two weeks early in pregnancy. Recommendations can then be made on the basis of this diet. The patient who has fruit juice, one egg and toast for breakfast, a leafy green salad, a vegetable of high color, a liberal serving of beef or other meat, an average amount of butter and one quart of milk (along with bread, desserts and other foods) will come within the suggested daily allowances of the nutrition experts.[6]

WHAT IS THE BEST FORM OF IRON TO USE?

The time-honored treatment of microcytic anemia of pregnancy has been the use of iron in one form or another, and the response to this therapy has been, to say the least, discouraging. An article published in 1948 by Talso and Dieckmann, who used ferrous sulfate, iron ammonium citrate or ferrous carbonate, reviewed the results in treatment of 250 cases of anemia of pregnancy. It was noted that at the time of their first visit 50 per cent of the patients were anemic. The conclusions drawn were as follows: "Two hundred and fifty cases of anemia in pregnancy were studied to evaluate the effects of treatment with iron alone and in combination with accessory factors. Controlled observations

indicate that the administration of these substances does not increase the rate of hemoglobin formations significantly."

It is extremely important to note that one year later, in March 1949, the senior author (Dieckmann[1]) published a similar study using a molybdenum-iron complex with quite a different conclusion: "We have never had other iron salts so advantageous in pregnant patients. Our results with the molybdenum-iron complex have been so striking that if the patient has taken this medication for three weeks and shows no significant increase in hemoglobin concentration, the therapy is stopped and a more extensive study (bone marrow biopsy, gastric analysis, reticulocyte count, etc.) made to determine the cause of the anemia." He stated further: "The increases in hemoglobin were so dramatic and so rapid that we postulated a possible decrease in the plasma volume. A few studies, however, have indicated that there was no decrease in the plasma volume, and the consistent and sustained increase in hemoglobin extending into months after delivery indicates even better than blood volume determination that there was an actual increase in the amount of hemoglobin."

We[7] have reported a study of 500 gravidas, selected consecutively from private practice. The incidence of hypochromic anemia was approximately 12 per cent; that of macrocytic anemia was 0.5 per cent. During the period of time covered by this study, the only antianemic product used was mol-iron, which is molybdenized iron as used in Dieckmann's report. We have not used any supplements in the form of vitamins or liver concentrates. In those cases where the patient did not respond to mol-iron within three weeks, a more complex deficiency was postulated and further studies were made. In the few cases of so-called macrocytic anemia which have come under our observation, we have not found it necessary to do bone marrow studies. However, these would be highly desirable in cases of refractory anemia. We have not encountered in private practice a case of true addisonian pernicious anemia, but we have the rule of thumb that any patient over the age of 35 who exhibits a macrocytic anemia should be suspected of true pernicious anemia. While recent studies would indicate that macrocytic anemia of pregnancy probably represents a true folic acid deficiency and will respond to folic acid alone, it has been our custom to give additional dietary supplements in these uncommon cases.

We would like, however, to point out that, in spite of the fact that the background for the anemia of pregnancy is still not thoroughly understood, it has been the experience of all investigators that vitamin supplements are of no value in increasing the rate of hemoglobin formation. If the physician feels that he is achieving this result with multivitamin therapy, he is, in our opinion, mistaken, and must justify the giving of these expensive dietary supplements on some other scientific speculation.

In macrocytic or megaloblastic anemia of pregnancy, the response to folic acid is nothing less than dramatic. Many of the cases reported in the literature were previously refractory to vitamin B_{12} and liver preparations.[3,4] The amazing rapidity with which the bone marrow, which has been in megaloblastic arrest, returns to normal is illustrated in the report of a case by Spies with a two year follow-up. Treatment consisted of 10 mg. of folic acid daily for 78 days, followed by adequate instruction in diet.

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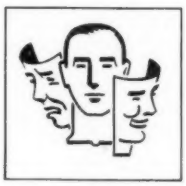
SCIENTIFIC EXHIBIT



Personality Manifestations in Psychosomatic Illness

VISUAL AID CHARTS TO PSYCHOTHERAPY

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THE charts which follow have been designed for the medical student and for physicians who seek an aid in getting the psychotherapeutic process under way. As the history is taken and the physical and laboratory examinations are made, both patient and physician are thinking of the cause of the complaints and the remedy to apply. In psychosomatic illness the remedy is rarely either surgical, chemical or other traditionally well known therapy, but consists of (a) understanding the emotional factors which are disturbing the physiology and (b) helping the patient to change them. Before being willing to work at modifying them, the patient must see and understand what they are as clearly as possible. It was to help in the process of further understanding through visualization that these charts were made. They can be of help when the presence of psychosomatic disease has been established and psychotherapy is beginning.

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ILLUSTRATIONS BY PHYLLIS ANDRE.

Color plates courtesy of Sandoz Pharmaceuticals.

SUGGESTIONS FOR USE

Chart 1, showing body organs and neural connections, has been found valuable to demonstrate to the patient that there are definite pathways which carry nerve impulses from the brain to all tissues controlling and modifying (1) muscle tonus, (2) blood supply and (3) gland activity.

Once this important phenomenon has been shown as a tangible cause or reason for local disturbance in an organ or system, the physician can pass to whichever chart pertains to the illness or organ under discussion.

The physician may say, "Here are some of the personality disturbances which have been found in other cases like yours. Do you think any of them may exist in you?"

The patient may answer in the affirmative

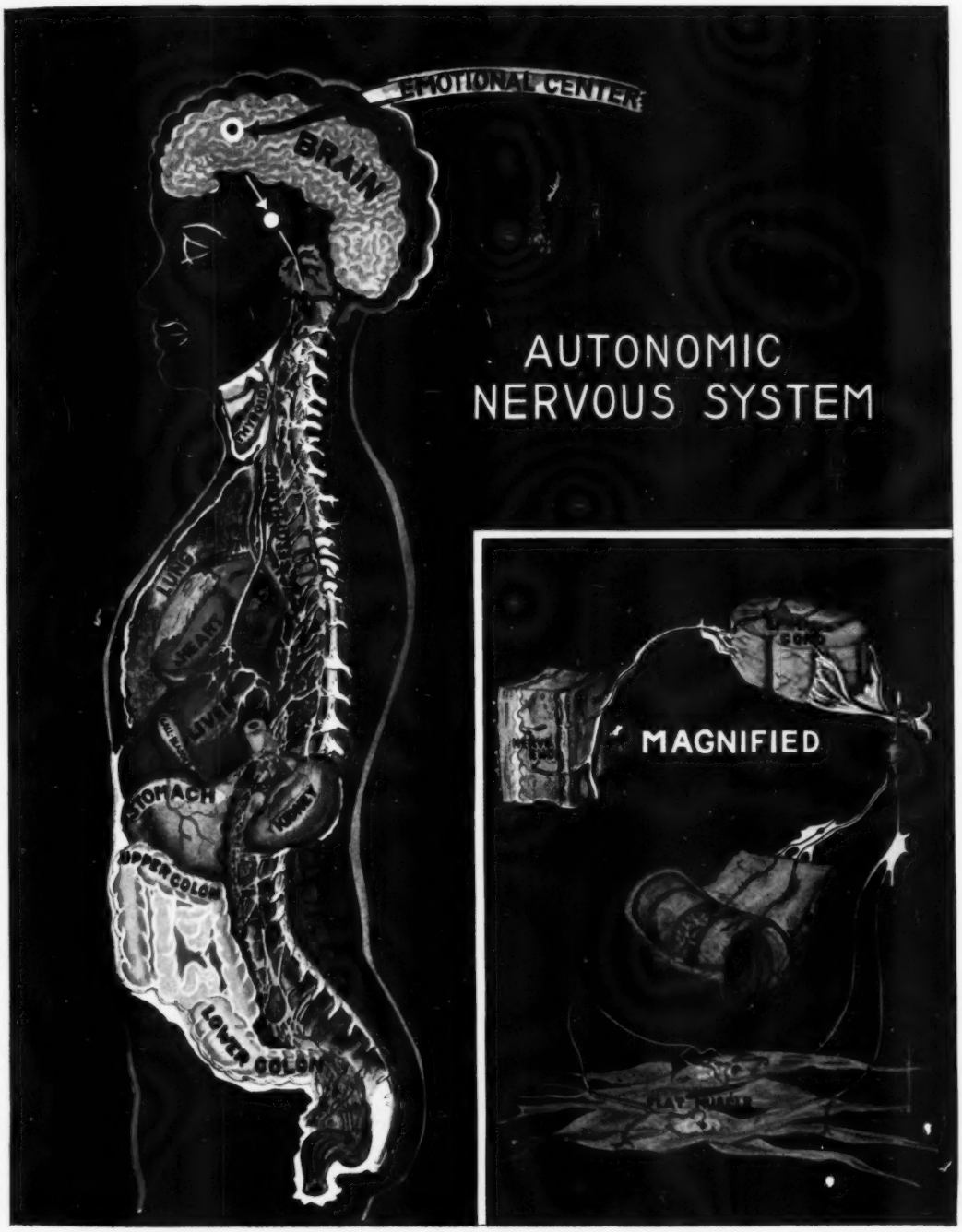
and proceed to indicate those he identifies as being part of his own make-up. Others may reply in the negative or express doubt, whereupon the physician can say, "Well, look again, study it a few minutes, meanwhile remaining as open-minded as you can." It is an unusually uncooperative patient who refuses to see anything in the charts for himself, provided (1) he has been given some explanation of the fact that emotions can cause symptoms and (2) the physician has been convincing in stating that, as a result of study of the case, disturbed emotions are the cause of the patient's difficulty.

It is suggested that the charts be used for as many sessions as are necessary for the patient to become familiar or "at home" with the idea that the personality discharges energy which finds its way through specific channels to disturb physiology and produce distress (or pain).

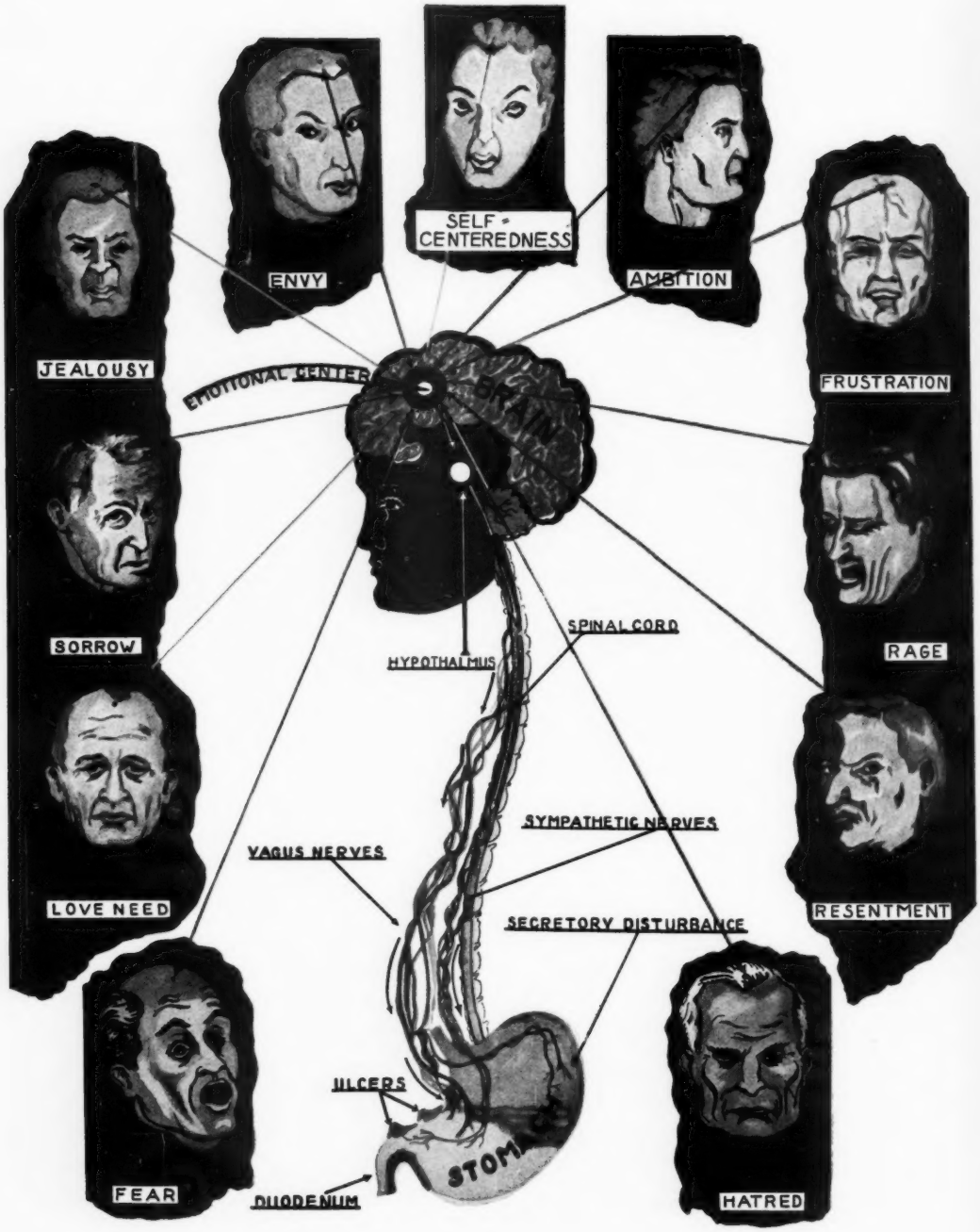
After the charts have served as an introduction to the psychotherapeutic process, one can proceed with efforts directed toward a discussion of conflicts, needs, tensions and their solutions. It is assumed that the physician has had some training and experience in psychotherapy. This can be supplemented by: "Psychotherapy in Medical Practice," by Maurice Levine, M.D., published by The Macmillan Company, New York. A typical case presentation is given following the illustrations.

CONDENSATION OF PSYCHOTHERAPEUTIC PROCESS IN PSYCHOSOMATIC DISEASE

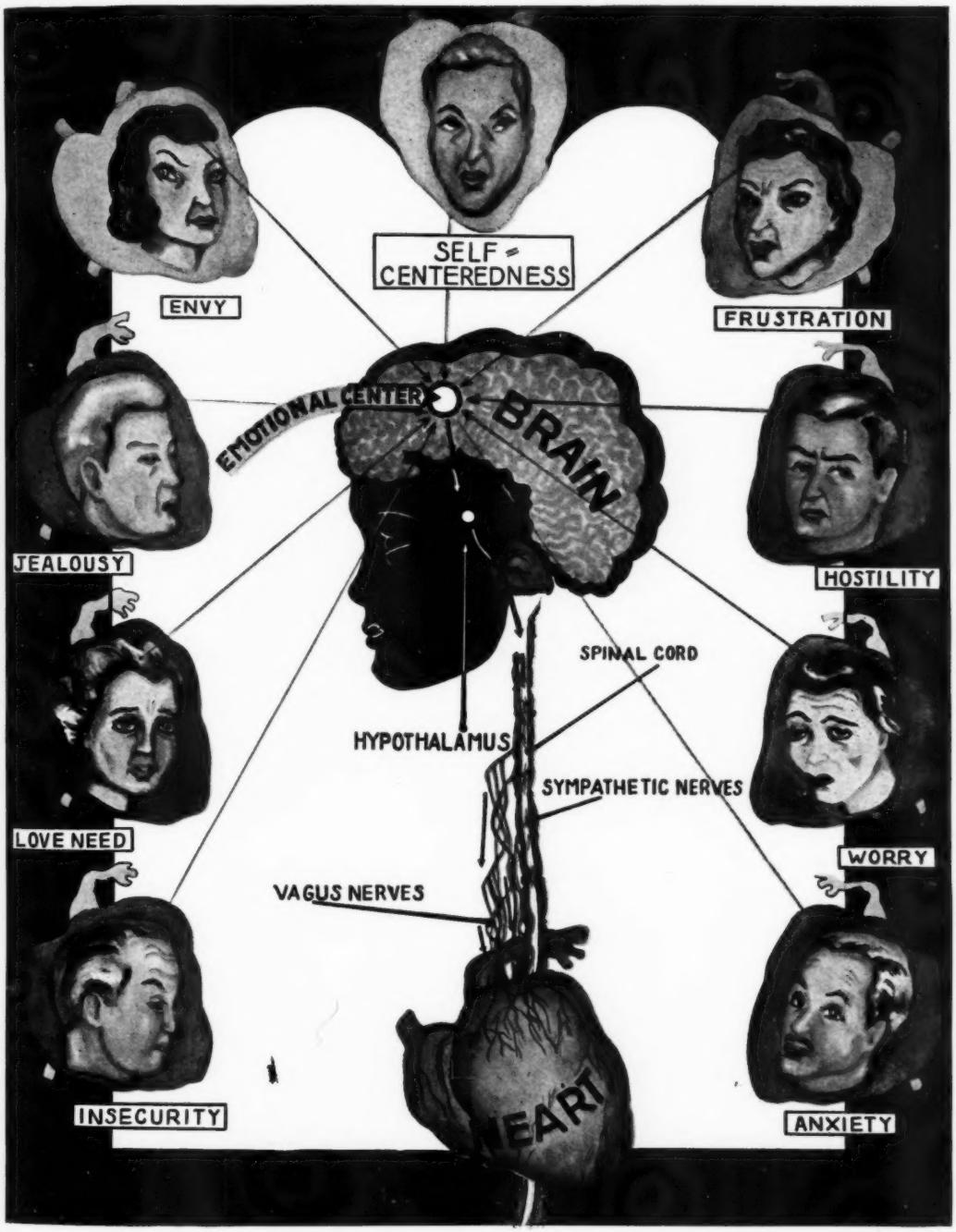
STATUS OF PATIENT	NEEDS FROM PHYSICIAN	MEANS OF ACCOMPLISHMENT
1. Possessor of distressing symptoms.	To be given explanation of symptoms which usually serves to neutralize fears of disease or disease results.	Example. Analogy. Charts. Pictures. Movies helpful in groups.
2. Possessor of personality with remnants of childhood emotions such as over-dependency, excessive love needs, resentment, hostility, envy, jealousy, etc.	Show that acceptance of hitherto unconscious emotions and finding an antidote for them will be productive of health through relieving effect of emotions on physiologic functioning.	Explain early life, effect of social training on physiology and the later effects of conflict and regression on body function.
3. Tends to avoid reality. Tends to avoid responsibility. Tends to protect self. Tends to indulge self. Tends to rationalize immature way of thinking and acting.	Teach patient a knowledge of human nature in general, through anecdote and analogy which aid him in understanding and accepting emotions in himself. Some need more direction and encouragement toward maturation than others.	Regular appointments. Faith in ability of patient to change. Try to see that patient receives some rewards for more mature behavior. Treat relatives if necessary.



NERVES: Pathways of emotional discharge through fibers to glands, blood vessels and muscles.



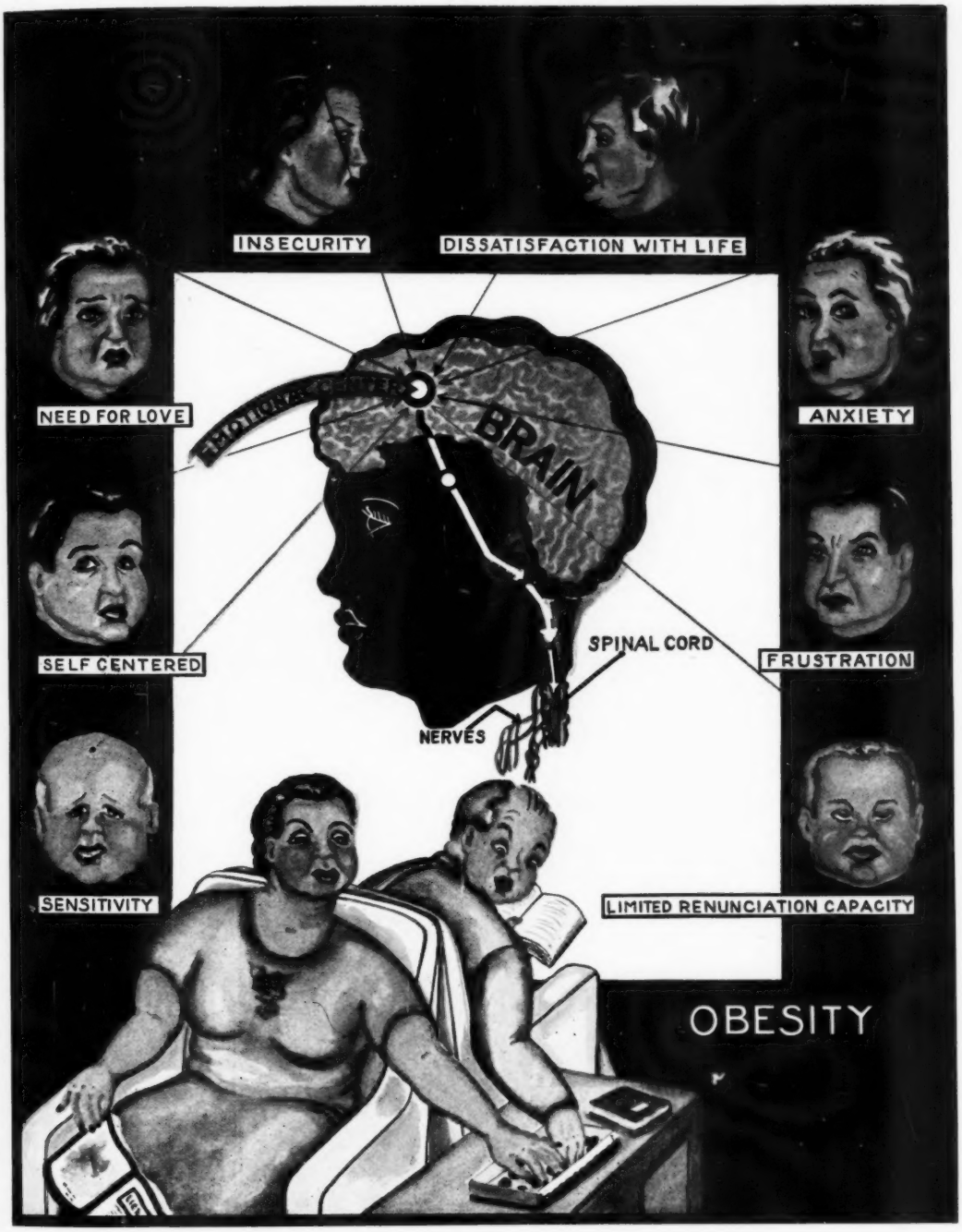
STOMACH: Emotions affect muscle tones, gland activity and blood supply of stomach.



HEART: Emotions can affect heart rate and rhythm.

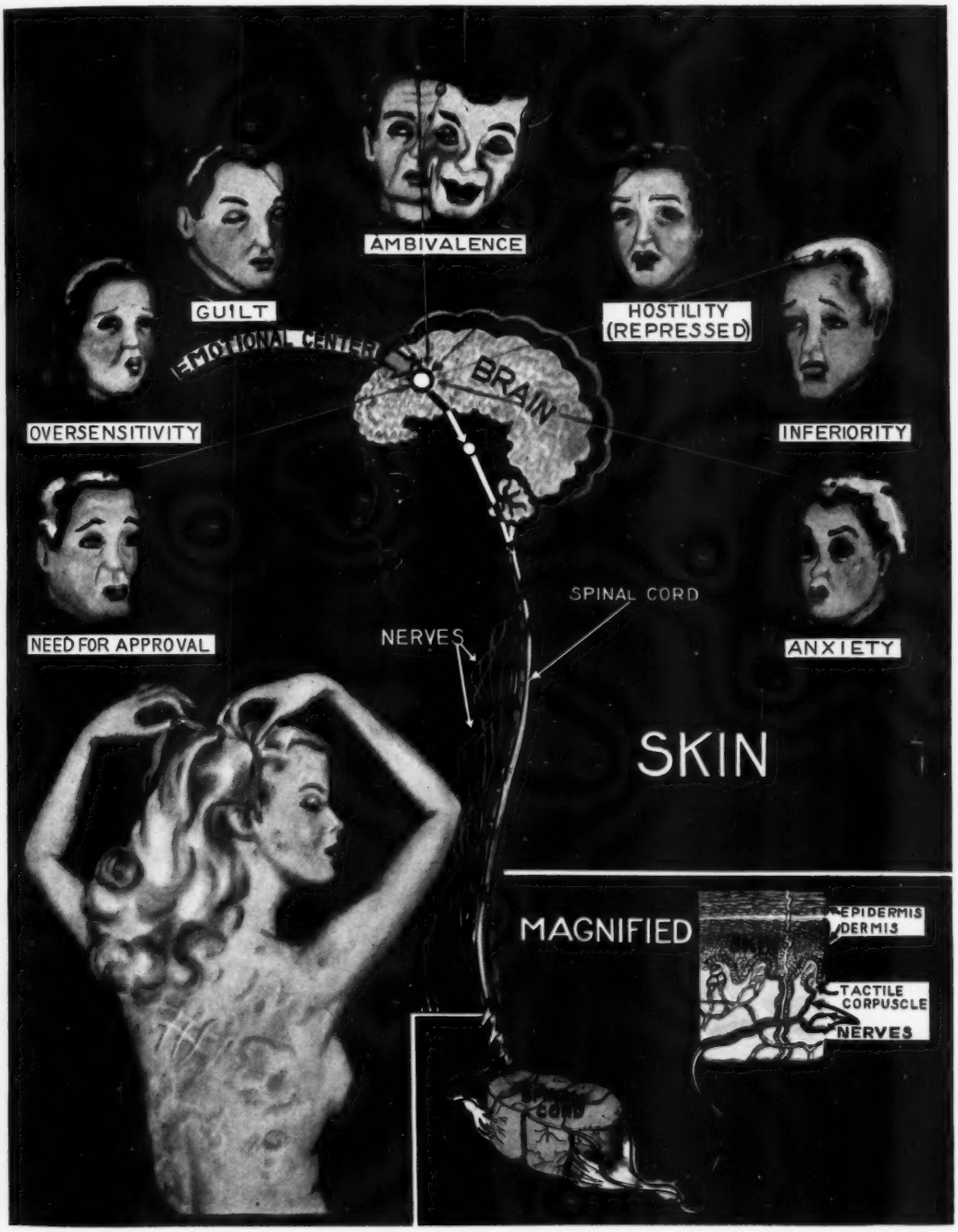
FATIGUE: Fatigue is produced by emotional stress and strain as well as by physical stress and strain.

Personality Manifestations in Psychosomatic Illness: English

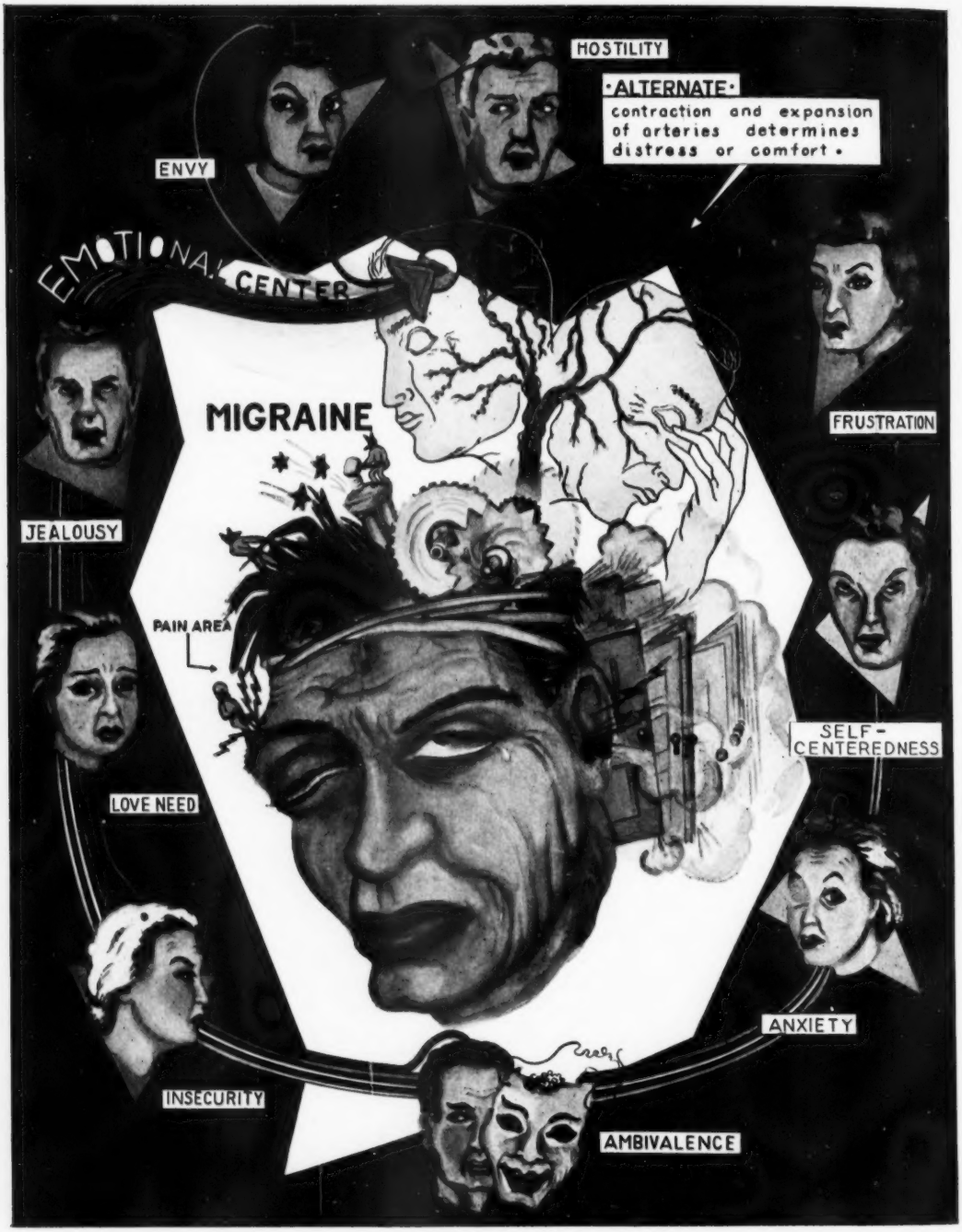


OBESITY: Eating may bring the gratification that is not obtained through healthy emotional outlets.

HEADACHE: Emotions frequently produce headache following changes in caliber of blood vessels.



SKIN: Emotions may adversely affect the healthy functioning of the skin.



MIGRAINE: Emotions can play a large role in producing migraine by periodically altering blood vessel caliber.

TYPICAL CASE PRESENTATION

Gastric Neurosis

Social and Psychologic History

INFANCY (age 1 to 2 years)—Mother pampered him. Father ignored him.

CHILDHOOD (age 2 to 6 years)—Timid, shy. No special interests.

GRADE SCHOOL—Fearful and tried to hide this by a limited participation in play.

HIGH SCHOOL (age 14 to 18 years)—A poor mixer. A few sports. Little interest in studies.

MARITAL (age 21 years)—Married gentle, competent woman in early twenties.

WORK—Went to work in small industrial plant where father had worked. Felt like "the kid," unaccepted; didn't know how to make friends. Could not share in camaraderie of the men.

AGE 25—Son born. While he was deriving so little emotional satisfaction from work, he was also ignoring the potential pleasures in enjoyment of his wife and child. His psyche was becoming impoverished and empty and life was more burdensome, formidable and anxiety-producing.

Symptomatology

Began to have anorexia in the morning and nausea after trying to eat. This was followed by distress after lunch and dinner—alternating constipation and diarrhea. A few weeks later he developed griping pain in the abdomen. After two months of symptomatic treatment he developed fatigue so that work became an additional burden. One month later he began to sleep poorly. Six weeks later he began to feel that the griping pains meant ulcer or cancer. He worked under great anxiety, saw many doctors and tried various self-medications, to no avail.

The unmet emotional needs for security, resulting in anxiety, were more and more disturbing to body physiology.

Careful physical examinations, x-ray and laboratory studies were negative. But the distress he suffered made him feel that something toxic or "sick-producing" was being overlooked. After 18 months of suffering, he stopped work and this event caused the treatment to be changed from antacids and sedation to psychotherapy.

PSYCHOTHERAPEUTIC PROCEDURE AND EFFECT ON SYMPTOMATOLOGY

PSYCHOTHERAPEUTIC PROCESS—1. Kept in foreground the examinations which revealed no tissue pathology.

2. Used charts to show autonomic nerve distribution to glands, blood vessels, muscle tissues and organs.

3. Called attention to emotions which disturb organ functions.

4. Pointed out his *need* for friendship and interest but also how he was missing it by *fear* of people and not knowing how to be friendly; being anxious about approaching people.

Effects

Some reduction of anxiety began when he received a tangible explanation for his symptoms. Doubts would creep in and he would ask, "Are you explaining the cause of* all *my trouble, as I feel 'wrecked' in my bowels?"

5. Gave him specific formulations of dynamics as learned from history as follows: "To get strength for healthy body-functioning again, you need security through better human relations. The men at work are willing to accept you if *you* accept them. Your wife and child need a cheerful man who will notice and appreciate them rather than one who worries about his digestion. Let your physician have the responsibility of thinking whether your body is sick or well. You relinquish that re-

sponsibility into trained hands while you do what you *can* learn to do; namely, to offer yourself to people in such a way that *they* can help cure you."

Effects

He would be reassured and then be able to turn his mind a little more in the direction of personality self-improvement. He slept better. He began playing golf with a little less fatigue. He protested, however, the idea of increased social activity. His wife liked dancing, while he did not, so he needed encouragement to learn. He hadn't played cards, so he learned—reluctantly, but he learned. He needed to be shown and convinced that a more altruistic and well rounded personality would be productive of better health.

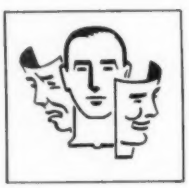
6. "Your body's distresses are secondary to your mind's need for love and good will and the sense of physical well-being which comes from good social relations. Take your wife and child out, go to the movies, invite your friends in. Begin that golf playing you have wanted to do. You do not need to suffer if you will begin new habits of thinking and acting."

Effects

But improvement in symptoms is always convincing and is one of the physician's best allies in psychotherapy.

The patient's upper and lower bowel quieted down; the griping pain stopped, appetite returned and digestion became comfortable. Three weeks after treatment began he was back at work, albeit with some residual symptoms, and in three months he was symptom-free.

7. "You need to live in friendly relations with people so that you make up to yourself for some of the lack of love and interest on the part of your father during your childhood. You should accept the fact that for health you need a wider range of interests and personal contacts for healthy living than either your mother or father required."



Pediatrics

IN GENERAL PRACTICE

For general practitioners, in whose practice is included a major proportion of the medical management of infants and children, and for the specializing pediatrician as well, POSTGRADUATE MEDICINE *presents this special regular department devoted to brief discussions by recognized authorities on their preferred methods of the treatment and management of diseases and problems of infancy and childhood.*

A Technic for Intravenous Therapy in Infants and Children

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INTRAVENOUS infusions have always been a problem in the care of infants and small children. Although satisfactory technics have been developed and are being used in a few pediatric centers, the great majority of practitioners throughout the country are still seeking substitutes for the intravenous route, or solutions to the technical problems of intravenous therapy in pediatric practice.

In an effort to eliminate some of the difficulties, I will describe in detail a method of catheterizing a peripheral vein with polyethylene tubing. When this is properly done, fluids can be administered continuously or intermittently for the duration of the illness by merely adding or changing bottles and regulating the speed of drip. In this series it was possible to continue intravenous support as long as was necessary in 94 of 100 consecutive cases, without changing the original venotomy. Moreover, the patient is more comfortable, free to move and can be easily transported for treatments, x-rays or conferences, without danger of disturbing the infusion.

The use of steel needles and cannulas in infants leaves much to be desired. The incidence of clotting in the needle and phlebitis of the vein is high. Needles frequently slip out or through a vein wall, or easily tear the vein wall.

Polyethylene was discovered in 1933 but was not used in the field of medicine until 1945. Its use for venoclysis has been described by several authors. Meyers,[1] in 1945, threaded tubing through a large-bore needle and left the tubing in the veins of adult patients as long as was necessary for repeated injection of intravenous medications. Guenther, Grindlay and Lundy,[2] in 1947, threaded polyethylene tubing through a 17 gauge intravenous needle into veins of adults for purposes of venoclysis and medication for periods as long as two weeks. Because this method could not be used in infants, whose veins are not large enough for such a needle, Alexander, Small and Campbell,[3] in 1948, dissected out the vein and directly inserted the tubing. They found this method satisfactory in

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90 cases. Farquhar and Lewis[4] reported a similar method in England about the same time. In 1949, Duffy[5] reported 72 cases in which polyethylene tubing was threaded through a needle into veins for venoclysis. In one case the tubing remained in the jugular vein for 39 days without complication.

Polyethylene is a plastic which is flexible, durable, chemically inert, light, nonirritating, fluid-repellent, easy to sterilize and inexpensive. Because of these properties its advantages are many. It repels fluids and therefore greatly delays the clotting of blood within its lumen. This is of special advantage in treating infants when fluids must be administered very slowly. Clotting can be prevented indefinitely if the tubing is rinsed occasionally with saline, as it lies in the vein when not in active use. The tubing does not react with surrounding tissues and thereby substantially reduces the incidence of thrombophlebitis. Its flexibility allows for movement of the vein during movement of the extremity. Rigid immobilization of the extremity, necessary when steel needles or cannulas are used, is thus eliminated. The patient is more comfortable and has much greater freedom of movement.

The polyethylene tubing is connected with the intravenous set by means of a standard needle or cannula which fits snugly into the lumen of the polyethylene tubing. Many sizes are available, but for treating infants we have used tubing of two different inside diameters: 0.86 mm. diameter accommodates a 20 gauge needle, and 1.14 mm. diameter accommodates an 18 gauge needle. Polyethylene tubing is sterilized by soaking cut lengths in 1:1000 aqueous ZEPHIRAN® solution for 24 hours. The tubing must be held under the surface of the solution in order to fill the lumen and prevent the tubing from floating.

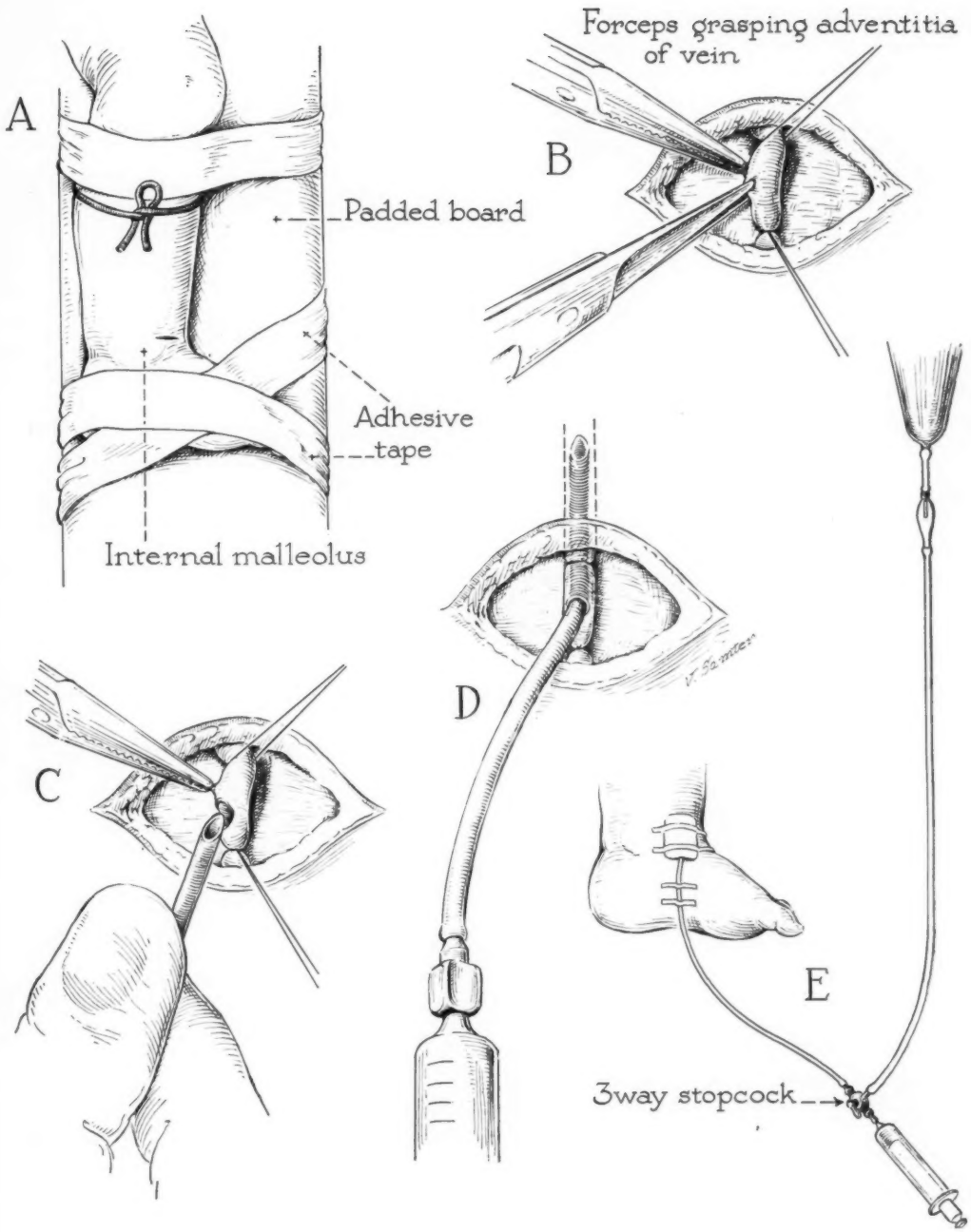
TECHNIC

Venous catheterization for venoclysis is a minor procedure, but at times it can be extremely difficult in an infant. A description of the technic in minute detail is therefore presented in an effort to eliminate some of the difficulties encountered.

The procedure should be done in a clean treatment room using sterile technic with gloves, masks and drapes. We prefer to use the saphenous vein where it lies constantly just anterior to the medial malleolus. The foot is immobilized in external rotation by binding it to a padded splint with adhesive tape. It is important to have a firm, fixed field when catheterizing a small vein. A second strip of tape is applied around the leg and splint above the field, but it must not be tight. (All immobilization material may be removed after the tubing is securely in place, for greater patient comfort.) The polyethylene tubing is then fitted with the proper needle on one end, and the other end is cut obliquely with a scissors. The tubing is washed, inside and out, with saline solution and its lumen is left full of saline. A tourniquet is applied to distend the vein and make it easier to locate. The skin is prepared and care is also taken to soak the nearby adhesive tape and padded splint with the preparing solution. Appropriate drapes are applied.

A solution of 1 per cent procaine is infiltrated just anterior to and at the upper level of the medial malleolus. A 1 cm. transverse incision is made with the tip of a pointed scalpel blade (Figure A). All layers of the skin must be cut or it will be difficult to separate the edges of the wound. With the opening motion of a small curved hemostat, the incision is spread apart. The spreading motions are continued in a direction parallel to the course of the vein until the vein is well exposed and freed from the underlying tissues. Sometimes, when the vein cannot be seen, it can be scooped up by sliding the curved hemostat under the tissues, where the vein is thought to lie, in a direction perpendicular to the course of the vein. When the vein is included in the tissues scooped up into the wound, it will lie on the hemostat as a flat, glistening, white ribbon. Using either of these two methods we have always been successful in finding the vein.

The vein is tied distally with a 5-0 silk ligature and a similar ligature is placed around the vein proximally but is not tied. Tension is made on both ligatures by the weight of a hemostat placed on the end of each ligature. Earlier in our series these ligatures were of 4-0 chromic gut. It was observed that they remained for long periods of time as lumps under the skin of the



Technic of insertion of polyethylene tubing in saphenous vein in ankle for intravenous therapy.

ankle and, not infrequently, even broke down and drained. This will happen especially in infants because the gut is so close to the overlying skin. We also found that black silk frequently showed through the skin. We now use 5-0 white silk for white infants and 5-0 black silk for Negro infants.

The tourniquet is now removed, having served its function. At this point it is important to have two very good instruments in order to complete the procedure with the least amount of difficulty. One is a fine-pointed hemostat which will hold a small piece of adventitia of the vein without slipping. This is applied. The second is a small scissors which will cut accurately at its tips. Holding the adventitia with the hemostat in one hand, the vein is partially cut in an oblique manner with the scissors in the other hand (Figure B).

Still controlling the vein with the hemostat on the adventitia, the polyethylene tubing is threaded into the vein for a distance of two to three inches. It must slide in easily; otherwise it is not in the lumen (Figure C). The proximal ligature is then tied around the vein and tubing and the hemostat removed from the adventitia (Figure D). One or two silk sutures are used to approximate the skin edges, and a small dressing is applied and secured with adhesive tape. The drapes are removed and the intravenous solutions are connected to the needle in the end of the polyethylene tubing. The needle and tubing are fastened to the skin with narrow strips of adhesive tape. The splint may now be removed (Figure E).

We find it convenient to connect the tubing to the solutions via a three-way stopcock so that the change between blood and other solutions may be made quickly and accurately with a minimum of mixing. Another advantage of such a connection is that blood can be pumped into the vein with a syringe, if necessary, and it is also useful for the administration of intravenous medications. When the third connection is not in use it may be sealed with a sterile syringe. If it is desirable to discontinue the fluids temporarily, the end of the needle or stopcock is sealed with a syringe half filled with saline. The syringe may then be taped to the outside of the leg and the patient need not be immobilized. If the fluids are to be discontinued for periods greater than four hours, the tubing should be flushed out with normal saline occasionally in order to keep the vein open.

DISCUSSION

Of 100 consecutive cases, 30 were infants less than 1 month old, 35 were between 1 month and 1 year old, and 35 were in the age group between 1 year and 11 years. The patients were carefully observed as to the length of time the tubing was allowed to remain in the vein, and for any complications developing as a result of the tubing. It was possible to give complete intravenous support to 94 out of the 100 patients, using the same intravenous catheter as long as was desired. The tubing was left in place for from 1 to 10 days. The average length of time per patient was three days. In 6 cases the tubing was removed prematurely. This was necessary once because the dressing became grossly contaminated with feces on the second day. One became plugged with a clot after three and one-half days. One had not been securely fastened and was inadvertently pulled out on the second day either by a nurse or by the patient. One was removed because of thrombophlebitis on the second day. In 2 instances removal was necessary on the second day because of wound infections.

There were 6 cases of thrombophlebitis in the series. One already has been mentioned, occurring on the second day and necessitating premature removal of the tubing. Three cases were noted on the third day and one on the fifth day when the tubing was removed, but intravenous therapy was no longer necessary. The sixth case occurred on the seventh day, one day after the tubing had been removed. In all instances the phlebitis subsided in a few days after removal of the tubing. Alexander, et al.[3] reported 8 cases of superficial phlebitis in 90 infants and children in whom polyethylene tubing was used. In 72 intubations of children and adults, Duffy reported a local reaction in 16 cases. In Anderson's study[6] of 62 patients receiving intravenous fluids from one to seven days through indwelling polyvinyl tubes, 23 developed thrombophlebitis.

There are several possible explanations for the

development of thrombophlebitis: (1) incomplete sterilization of the tubing; (2) failure to wash the sterilizing solution from the tubing; (3) trauma to the vein wall by movement of the indwelling catheter; (4) trauma to the vein wall by the pressure of too large a catheter; (5) the use of irritative intravenous solutions such as protein hydrolysates, hypertonic solutions and aureomycin, and (6) the factor of age, because thrombophlebitis appears more frequently in adult patients.

Tuell, Martin and Laufman,[7] in addition to discussing thrombophlebitis, mentioned two other complications. One of these is the occurrence of localized edema at the site of venotomy. We noticed this occasionally but did not consider it an indication for removal of the tubing. The other complication described by these authors occurred in a dog on whom the tubing was not securely fastened. Between infusions, the tubing was drawn into the vein and carried centrally as a fatal embolus.

SUMMARY

1. The importance of a reliable and convenient technic for intravenous infusion in infants and small children is stressed.

2. Polyethylene tubing has many advantages over steel needles and cannulas.

3. A detailed technic for venous catheterization with polyethylene tubing is described.

4. Observations on 100 consecutive cases are presented and the complications are discussed.

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"Accidental" Ingestion of Poisons in Childhood

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Poisoning continues to take its annual toll of children, principally among those from 1 to 4 years of age—the time when locomotion and curiosity far outweigh knowledge and judgment. The success in combating other hazards has raised accidental deaths to first place in mortality rates for this age group. The small but significant percentage contributed by poisons does not reveal the probably far greater morbidity.[1,2] Barbiturates, kerosene, salicylates, arsenic, strychnine and lye appear to be the Big Six in pediatric poisoning.

These poisonings are generally considered accidental, by legal definition, "an unexpected happening not due to any negligence or malfeasance."[3] An accident is an accusation of negligence, however, when defined as "an event that takes place without one's foresight or expectation"[3] for physicians can only expect that the toddler learning by exploration may ingest any poisonous substance that foresight has not made less attractive or placed beyond reach.

The prevention of poisoning requires protection and education. Although protection is paramount in the age group most frequently involved, effective educational experiences adapt-

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ed to the child's level of development may further decrease the risk.[4] Parents can profit even more from education. The physician's educational use of a home check-list of poison hazards may succeed with those deaf to "all medicines in a locked closet, no inflammable liquids and no cleaning or polishing materials on lower shelves or floor, and no pennywise saving of solvents and insecticides in pop bottles." We are culpable also in our tacit approval of many home remedies of dubious worth.

The pediatric toll due to poisonings can be reduced through emphasis on their nonaccidental nature and continuous doctor-parent and parent-child education.

DIAGNOSIS OF POISONING

Poisoning must be considered in any unexplained illness, particularly that of an acute nature. The signs and symptoms may be suggestive but are not diagnostic. Clues such as the garlic odor associated with phosphorus ingestion, the persistent chocolate hue of venous blood in methemoglobinemia, or the odor of oil of bitter almond found in cyanide and nitrobenzene poisoning are all too infrequent.

Knowledge of the sources, actions and treatment of every known poison is desirable. Command of the general principles of management, however, is even more desirable, for too often the next poisoning encountered will be due to a new or not readily identifiable substance. The general principles are: identification of the poison, prevention of further absorption, use of physiologic antidotes or treatment of the hazardous manifestations, and the provision of fluids, calories, electrolytes, vitamins, etc., to cover not only normal daily needs but also those demands increased by the metabolism or elimination of the poison.

IDENTIFICATION OF THE POISON

In poisoning, known or suspected, the immediate problem is twofold: (1) to identify the poison and (2) to prevent further absorption. The household source of the substance ingested, its intended use and a call or telegram to the manufacturer may lead to the identification of the poison. The container, stomach contents (emesis or lavage) and the urine should be saved for analysis. Prompt identification of the poison may permit valuable anticipatory measures even though specific chemical or physiologic antidotes are not known.

TABLE 1
CHEMICAL ANTIDOTES

POISON	CHEMICAL ANTIDOTE
Phosphorus	Copper sulfate, 0.2%, 1 gm. per 500 cc. water
Fluoride	Calcium chloride, 5%
Mercury bichloride	Sodium formaldehyde sulfoxylate, 5%
Phenol	Olive oil
Lye and other caustics	Weak acids (vinegar, lemon juice)
Acids	Weak alkalis (milk of magnesia, baking soda)
Formaldehyde	Dilute ammonia water, 0.2%
General	Activated charcoal U.S.P.

PREVENTION OF ABSORPTION

Absorption of the poison may be minimized by emesis, measures to delay gastric emptying, gastric aspiration and lavage, the introduction of a specific or general chemical antidote, and the use of cathartics. Emetics, though time-honored, are too apt to cause aspiration of vomitus in the age group most frequently ingesting poisons. Emetics are contraindicated in coma and for kerosene poisoning. Many poisons induce vomiting by local irritant action. Where this does not occur it might be better to have the child drink milk.[5] Milk is available. It is known to prolong the gastric emptying time, thus delaying the absorption of alkaloids. Milk aids in precipitating metallic poisons. Its calcium content will convert sodium fluoride to nontoxic calcium fluoride. In addition, with the possible exception of furthering phosphorus absorption, whole milk lacks the dangers inherent in many measures recommended.

Gastric aspiration and lavage may be life saving when properly performed. As frequently done, however, gastric lavage is more hazardous than helpful. It engenders a false sense of accomplishment. The usual lavage hastens the passage of gastric contents. It increases the likelihood of aspiration into the lungs—most likely in toddlers and comatose subjects. Finally, lavage may induce fatal seizures in convulsant poisoning.

TABLE 2

PHYSIOLOGIC ANTIDOTES

POISON	ANTIDOTE	USE
Arsenic, mercury, bismuth, gold, antimony	DIMERCAPROL® (BAL) 10% solution in peanut oil	3 to 5 mg. per kilogram every 3 to 4 hours, intramuscularly.[8]
Nitrites, aniline dyes, chlorates, etc., producing methemoglobinemia	Methylene blue 1% aqueous solution	1 to 2 mg. per kilogram intramuscularly, over 5 minutes. 3 to 5 mg. per kilogram orally, acts more slowly.[9]
Cyanide	Amyl nitrite, sodium nitrite and sodium thiosulfate	Amyl nitrite by inhalation pending intravenous sodium nitrite (0.3 to 0.5 gm. as 3% solution at 2.5 to 5.0 cc. per minute) followed by sodium thiosulfate intravenously (12.5 gm. as 25% solution over 10 to 15 minutes).[10]
Sodium fluoroacetate ("1080")	Monoacetin (glycerol monacetate)	0.1 to 0.5 cc. per kilogram intramuscularly.[11]
Organic phosphate insecticides (Parathion, TEPP, HETP)	Atropine (against muscarine effect)	Large doses tolerated, given intramuscularly, hourly until signs of atropine reaction. 0.3 to 0.6 mg. for a 14 kg. child.[12]

Gastric aspiration-lavage technic—The child should be restrained, placed with his face to one side and lower than the chest, and either adhesive tape-wrapped tongue blades or a mouth gag inserted. By means of a large-bore tube (28 F or larger) passed into the stomach, the gastric contents are aspirated with an Ewald aspirating bulb.[6] Then lavage should be performed, injecting and aspirating small amounts (150 to 250 cc.) repeatedly, using a total of two or more liters of fluid. The use of large volumes ignores the established principle of greater recovery with repeated small washings, and also promotes passage into the intestine.[7] A urethral catheter and a 50 cc. glass syringe may, of necessity, be employed. They are not recommended. A urethral catheter, or similar gauge polyethylene tubing with larger bore, does not permit the removal of pieces of poison or poison-impregnated material. Lavage is *contraindicated* in convulsions and after ingestion of corrosive poisons. It requires meticulous care to prevent aspiration in comatose or stuporous children and in cases of kerosene ingestion.

Tap water is generally satisfactory for lavage. Its efficiency is enhanced by the addition of activated charcoal U.S.P. (1 to 2 tablespoons, 10 to 20 gm.). The exceptions, with their specific chemical antidotes for initial lavage, are listed in Table 1. It is not clear that potassium permanganate, tannic solutions or "universal antidote" will accomplish more than the measures already described. Before the lavage tube is withdrawn, additional charcoal in water with a saline cathartic (magnesium sulfate, 2 to 4 gm.) may be left in the stomach to hasten evacuation of poison not recovered. Catharsis is not advisable for phosphorus, arsenic and other poisons inducing dehydration and shock.

PHYSIOLOGIC ANTIDOTES

The effects of the poison may be counteracted by a physiologic antidote (Table 2). Where such is not available and the poison is known, extreme deviations from homeostasis may be prevented by anticipatory therapy. Even though children may consume unpalatable poisons in large and lethal amounts, much more often the toddler has ingested just enough to alarm the parents. After measures to prevent absorption, watchful waiting is better than naive therapy, which in itself may require counter-therapy.

TREATMENT OF HAZARDOUS MANIFESTATIONS

Beyond the provision of maintenance needs, only dangerous manifestations should be treated. Shock, coma, cyanosis (anoxia), convulsions, acute urinary suppression and severe acidosis are the hazards most prejudicial to the child's recovery. The quantities and dosages in the following paragraphs are applicable to a 2½ year old child.

Shock may complicate many poisonings. It

should be anticipated in phosphorus, mercury bichloride, phenol, fluoride, arsenic and severe barbiturate intoxication. The treatment is the same as in the adult, but the amounts per kilogram of body weight are greater. Compatible whole blood should be given as soon as possible, meanwhile using isotonic sodium chloride or plasma intravenously. The volume may be estimated on the basis of 20 to 30 cc. per kilogram of body weight.

Coma may represent primary depression by barbiturates, opiates, hydrocarbons, etc., or be due to anoxia produced by carbon monoxide, cyanide, methemoglobinemia or respiratory insufficiency. The deeply comatose child may be compared to the surgical patient hovering between the third and fourth stages of anesthesia. Although the impression persists that the use of stimulants to lighten central depression should command first attention, the major problem is to maintain adequate respiratory exchange and an effective circulation.

In a recently reported series of severe barbiturate poisonings an unusually low mortality has been reported.[13] No analeptics were used. Emphasis was placed on maintenance of adequate respiratory exchange and circulation. Maintenance of adequate respiratory exchange requires oxygen therapy and maintenance of an airway. There must be provision for artificial respiration if the normal bellows action of chest and diaphragm fail. Constant attention is necessary to prevent the deleterious effects of accumulated secretions. This is attained by frequent changes in the patient's position, elevation of the foot of the bed and mechanical aspiration of pooled secretions. Since anoxia raises the threshold of the respiratory center, oxygen should be used before rather than after respiration has failed. Although respiratory stimulants may be used, they may not be necessary if the other means of maintaining oxygen-carbon dioxide exchange have been employed.

Hypotension may be a problem, particularly in barbiturate poisoning, either as a result of anoxia or from excessive doses of the drug. Where this occurs, the use of a vasopressor drug may be of value (ephedrine, 5 to 10 mg. or NEO-SYNEPHRINE®, 0.5 to 2.0 mg. every 30 minutes). Hypotension may give way to shock in severe anoxia. Efforts to decrease central depression have centered about the pros and cons of analeptic therapy. Picrotoxin is currently most widely used. Analeptic drugs are contraindicated in coma due to opiates, where anoxia is the major factor. Patients who still manifest a knee jerk and corneal reflex and who show a change in respiration subsequent to stimulation, appear to survive without the administration of picrotoxin. In those with areflexia, picrotoxin (0.3 per cent solution) may be of value, 5 mg. given intravenously, very slowly, every 15 to 30 minutes until the appearance of signs of excitation, finger-flicking, grimacing or spontaneous movements of the extremities.[14] Amphetamine as BENZEDRINE® (10 to 20 mg.) or DEXEDRINE® (5 to 10 mg.) every 15 to 60 minutes is a useful adjuvant.[15] It is a powerful cerebral stimulant without inducing convulsions. Amphetamine increases the respiratory minute volume and, at first, produces an increase in the blood pressure.[16] The initial doses may be given intravenously; subsequent doses, intramuscularly.

Cyanosis should be considered an indication of serious anoxia. The occurrence of anoxia without cyanosis or of methemoglobinemic cyanosis without severe anoxia should be appreciated. The measures to combat anoxic anoxia were presented in the management of coma. Failure of these measures to relieve cyanosis should suggest methemoglobinemia. This may be confirmed spectroscopically or by the failure of oxalated venous blood to become bright red when shaken in air for 15 minutes. Cyanosis may also be a terminal manifestation in cyanide poisoning. See Table 2.

Convulsions may result from stimulation (e.g., strychnine, camphor), hypocalcemia or anoxia. Short acting barbiturates should be used for stimulant convulsions. Up to 150 to 200 mg. of SODIUM AMYTAL® or pentobarbital sodium may be given slowly, intravenously, as a 5 per cent solution. Ether, ethyl chloride or chloroform may be used by open drop method pending preparation of the barbiturate.

Hypocalcemic manifestations, including tetanic convulsions, may occur in poisoning by fluorides, oxalates and DDT® (dichloro-diphenyl-

trichloro-ethane). This tetany may be controlled by calcium therapy. Calcium chloride, 10 per cent solution diluted to 1 per cent with normal saline, may be given intravenously, slowly, in a dosage of 0.5 to 1.0 gm.[17] The effect is transient. Calcium therapy should be continued orally as 10 per cent calcium chloride diluted to 2 to 3 per cent with milk or other fluids, giving 3 gm. or more daily in divided doses.

Acute urinary suppression, in poisoning, may follow shock or the action of nephrotoxic poisons (e.g., mercury, bismuth, carbon tetrachloride). Conservative therapy appears to have a higher survival rate than more energetic therapy. The maintenance of fluid balance is achieved by giving only sufficient fluid to maintain a constant daily body weight. The fluid to maintain body weight should be given as nonelectrolyte solution (5 to 10 per cent glucose in water). Additional fluid may be required to replace that lost by vomiting and diarrhea. The amount equivalent to urine and emesis volume is given as equal parts isotonic sodium chloride and 10 per cent dextrose in water. The administration of carbohydrate as glucose (50 gm. or more daily) will minimize protein catabolism and ketosis. Gradual resumption of renal function can be expected after 7 to 14 days, if the patient has not been drowned by excessive fluid therapy meanwhile. With the onset of diuresis, salt and water loss may be abnormal. At this time new imbalances may require replacement therapy.

Severe metabolic acidosis may occur in salicylate poisoning after the initial respiratory alkalosis. This is particularly true in younger children in whom coincident infections, anorexia and depletion of glycogen stores may be contributory. Severe acidosis due to formic acid occurs in methanol and formaldehyde poisoning. The provision of sufficient water, glucose and sodium will require their parenteral administration. Base may be supplied as sodium lactate or sodium bicarbonate; 30 cc. of sixth-molar lactate or 1.5 per cent sodium bicarbonate solution per kilogram of body weight increases the serum carbon dioxide value approximately 9 mEq. per liter or 20 volumes per cent. Although large quantities of sodium may be required over a period of days, greater than 70 cc. of sixth-molar lactate per kilogram of body weight should be avoided over short periods because of the danger of alkalosis and tetany.

After the initial restoration of renal output and partial correction of the severe acidosis, fluids by mouth are preferable to reliance on parenteral therapy exclusively. The daily provision in a 30 month old child of one liter or more of 5 per cent glucose in water will aid in preventing ketosis and achieving maximal protein sparing. The theoretical and practical aspects of the use of potassium-containing fluids and fluid therapy in general are presented in detail by Darrow and Pratt.[18]

The metabolism or excretion of poisons obviously superimposes added requirements. Failure to include the daily basal nutritional needs in the therapeutic program may nullify all the measures presented. The provision of ascorbic acid and the B complex vitamins should not be forgotten because of the emphasis which is given other needs.

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EDITORIALS

MARCH 1952

POISONING ACCIDENTS AND THEIR PREVENTION*

APPROXIMATELY one thousand deaths due to accidental poisoning in children are recorded annually in this country. The actual number is probably much greater, since many fatalities due to poisoning are attributed to other causes. In 1949, there were 70 deaths in North Carolina from accidental poisoning; 23 were in children (0 to 14 years).

Lye	7	(all under 2 years)
Kerosene	5	
Alcohol	2	
Salicylates (sodium and methyl)	4	
Arsenic	1	
Barbiturates	1	
Strychnine	1	
Sulfonamide drug	1	
Sodium fluoride	1	

For comparison, there were 22 deaths from poliomyelitis in 1949 in all age groups. Poliomyelitis is singled out in particular, for, in North Carolina, as in most states, there exists an almost mass hysteria throughout each "polio season." Yet no one gets excited about the numerous accident fatalities.

Mortality rates, however, do not adequately indicate the incidence of child poisoning. Figures from the leading hospitals in North Carolina give an incidence of approximately 150 children with caustic alkali (lye) poisoning each year. The incidence for kerosene poisoning would be several times the above figure if accurate figures could be obtained. In a four year study of admissions to Children's Hospital, Washington, D. C., there were 250 cases of accidental poisoning with 2 deaths.

Even though these figures may not seem impressive to many, it must be realized that many children who survive the ingestion of poison may be left with permanent disabilities, as for example, the esophageal strictures that follow the ingestion of caustic alkali (lye) and the hepatic or renal damage caused by many other toxic agents. One welfare agent found that the cost of surgical treatment and hospitalization over a period of time for just one indigent child with a stricture of the esophagus amounted to $8,000.

Poisons contained in household agents are responsible for most of these deaths and disabilities. If all drugs and household agents that contain poison were made inaccessible to children, such poisonings could be almost entirely prevented. The number of children who have been accidentally poisoned as a result of parental carelessness is truly tragic.

Since most infants and children are poisoned in or near the home by these agents, at least two preventive measures should be

*See *Pediatrics in General Practice*, page 239, this issue, for further discussion on this subject.

attempted: First, parents should be informed and educated to the dangers that lie ahead for their infant. As Dietrich[1] so aptly puts it, they must be shown that pimples and pounds are relatively unimportant problems. Accidents present a greater hazard than the threats our too thoroughly propagandized society ordinarily fears for its children. This can be accomplished in part by offering an accident handbook at the first year check-up with a list of various household agents containing poisons. Another measure would be more stringent federal or state laws.

The Federal Caustic Poison Act enforced by the Food and Drug Administration requires that the word "Poison" and an acceptable antidote be placed prominently on the label of a household package of a preparation which contains more than a specified amount of any one of the following ingredients: hydrochloric acid, sulfuric acid, nitric acid, carbolic acid, oxalic acid, any salt of oxalic acid, acetic acid, hypochlorous acid, potassium hydroxide, sodium hydroxide, silver nitrate and ammonia.

The Federal Food, Drug and Cosmetic Act, also enforced by the Food and Drug Administration, does not require the use of the poison legend on any drug preparation.

The Federal Insecticide, Fungicide and Rodenticide Act of 1947, enforced by the Department of Agriculture, requires that certain white economic poisons be colored, and requires that the labels of economic poisons toxic to man bear the skull and crossbones and the word "Poison" prominently in red on a background of contrasting color with a statement of the antidote for the economic poison.

These Federal Acts are fine and useful as far as they go; however, they are far from adequate. To an inquiry about more comprehensive federal laws the following reply was received from Dr. Erwin E. Nelson, Medical Director of the Federal Security Agency:

> "We are aware of the needless tragedies caused by accidental poisonings in cases where the poisonous article is not packaged or labeled in such a manner as to call attention to its poisonous properties. When a bill was introduced into the Senate in 1949 (S. 2193, 81st Congress) to require distinctive containers for certain poisonous drugs, we expressed our support of the purpose of the bill. We suggested that the Federal Caustic Poison Act should be broadened to cover poisonous substances other than those now specified in it, including drugs, and that the Federal Security Administrator be empowered, after opportunity for hearing, to add poisonous substances to the list; that provision be made for distinctive safety containers, distinctive coloring of the article where required and, in the case of poisonous liquids which might intrigue children, bottles with openings sufficiently constricted to make it unlikely that a child would ingest a lethal quantity.
>
> "S. 2193 was not passed by the 81st Congress and, so far as we know, no similar bill has been introduced into the 82nd Congress."

The medical profession should see to it that their elected representatives in Congress are made aware of the importance of more stringent federal laws concerning the packaging and labeling of poisonous articles or agents.

JAY M. ARENA
Duke University School of Medicine and Duke Hospital, Durham, North Carolina

1. Dietrich, H. F.: Accidents, childhood's greatest physical threat, are preventable. J.A.M.A. 144:1174 (December 2) 1950.

EXPENDITURES FOR MEDICAL CARE

In England following the enactment of the National Health Act, great numbers of people poured into doctors' offices with all sorts of trivial complaints. As a result, doctors were overwhelmed and were unable to give to the individual case anywhere near the amount of time usually given in the United States to individual patients by their physicians.

From England reports indicate that many patients now postpone going to the doctor's office because of the length of time required in waiting to see the physician. A recent report of the Sloane Foundation, published in *Fortune* for December 1951, calls attention to the deterioration in the quality of medical service that has occurred. The report says: "The Health Service, in its pres-

ent form, has proved immensely costly and has involved much waste. In the long run the heaviest cost may well be the serious deterioration in the standards and the efficiency of the medical profession itself." And again, "The doctor must inevitably see everybody—because any case may be a critical case. The queue, however, is so much lengthened by the presence of frivolous cases, encouraged by the fact that the service is free, that everybody gets less satisfactory treatment."

Most recent of the reports of Frank B. Dickinson, Ph.D., of the American Medical Association's Bureau of Medical Economic Research is devoted to medical care expenditures, prices and quantity for the period 1930 to 1950. This carefully documented analysis establishes again that the largest consumer expenditures were for food and tobacco. The other groups for which expenditures are large include clothing, accessories and jewelry, housing, household operation and transportation. The lowest expenditures were for private education and research, and medical care and death expenses constituted 4.9 per cent of the consumer's budget. By comparison, 4.2 per cent was spent on alcoholic beverages, 5.8 per cent for recreation, 2.3 per cent for tobacco and 1.2 per cent for personal care. The consumer has a choice, and he may devote such portions of his budget as he chooses to medical care, taking from other expenditures in order to meet these costs. Incidentally while expenditures for other purposes have risen almost continuously since 1930, the proportion of the budget spent for medical care has remained rather definitely around 4 per cent.

Over a 20 year period, the physician's average share of the dollar spent for medical care fell 12 per cent whereas the hospital share rose 66 per cent. The dentist's share fell 26 per cent whereas the portion spent on drugs and sundries fell 12 per cent. The rise in the cost of hospital care obviously reflects the greatly improved quality of hospital service brought about by standardization at a high level, the increased use of the hospital for teaching purposes and coincidentally, of course, the rise in payment of workers. These problems are now to be given special attention by the Commission on the Financing of Hospital Services and the analysis made in a two year study, including some experimentation, may yield important information as to how hospital costs may be lowered.

The quality of medical care in the United States has improved tremendously as measured by the increase in life expectancy at birth, the lessened amount of illness, and the shortened duration of stay of the patient in the hospital. These are values difficult to express in terms of dollars but easily appreciable by the people who have gained these values. Psychologically the indifference of many Americans to the importance of providing for medical services is due to the fact that so few of them now find it necessary to seek the aid of the physician or the services of the hospital in comparison with the amount of illness and fear of ill health and death that formerly prevailed.

MORRIS FISHBEIN

THE DECLINE OF ERYSIPELAS

When specific therapeutic measures are generally applied and truly effective, their results should be widespread and reflected nationally on the mortality of a disease. In the latest revision of Brenneman's "Practice of Pediatrics,"* Dr. Jean V. Cooke points out how erysipelas illustrates such success to a striking degree. Treatment with sulfonamides and antibiotics has brought about a most amazing change not only in the prognosis and course of erysipelas, but also in its incidence. Figures from the vital statistics of the United States Bureau of the Census for the years 1925 through 1947 vividly demonstrate the point. Before 1937 well over 2000 deaths occurred annually. Coincidentally with the introduction of sulfonamide drugs in 1937, there was a rapid and continuous fall in the number of deaths to less than 5 per cent of the previous

*Brenneman-McQuarrie: "Practice of Pediatrics." 1951, W. F. Prior Company, Inc., Hagerstown, Maryland.

level, the actual number in the latest available report (1947) being only 82. Of special interest is the decrease in mortality of infants under one year of age, the largest proportion in the neonatal period. Deaths from erysipelas in this group, which had ranged from 500 to 700 annually previous to 1937, have now fallen to less than 2 per cent of this figure. Only 12 deaths were reported in 1947.

Obviously mortality is closely related to morbidity; when fewer cases of a disease occur, fewer deaths are to be expected. However, it is now apparent that the decrease in the incidence of clinical erysipelas is to be credited to the sulfonamide and antibiotic therapy used on patients with streptococcal infection. Responding favorably to such treatment, they have not developed clinical erysipelas, or in the few instances in which it has appeared it has been a minor and temporary illness with little danger to life. Studies cited by Dr. Cooke indicate that a possible reason for its virtual disappearance is bound up with the nature of the infection. Idiopathic erysipelas is usually a secondary skin inoculation of streptococcal disease already present in the upper respiratory tract. The frequent eruption of erysipelas around the face and head further supports this view.

Nowadays prompt use of sulfonamide and antibiotic therapy in patients with streptococcal disease has succeeded in preventing, for the most part, development of clinical erysipelas. Actually the incidence at present is so low that it is difficult for one unfamiliar with the pre-sulfonamide era to secure sufficient experience with the disease to discuss its distinctive features. Similarly, in the early years of sulfonamide therapy, reports of its effectiveness on erysipelas quickly appeared, but in more recent years no references have appeared, apparently because of the near-complete control of the disease by these modern drugs and the related decrease in incidence.

THE UNITED STATES ARMED FORCES MEDICAL JOURNAL

One of the outstanding evidences of the effectiveness of cooperation in activities of the Army, Navy, Air Force and the Department of Defense is the publication of the United States *Armed Forces Medical Journal** under the joint editorship of two colonels and a captain, Drs. Wayne G. Brandstadt of the Army, Robert J. Benford of the Air Force and William R. Whiteford of the Navy. This publication is of high quality in the field of medical periodicals and the articles in any issue afford much important medical information.

In a preliminary paragraph Dr. W. Randolph Lovelace, chairman of the Armed Forces Medical Policy Council, indicates some of the advancement that has taken place through the Council during the past year. Included are the standardization of medical recording and reporting procedures, the establishment of the Armed Forces hospital expansion planning program, the approval of preliminary drawings of standard plans for permanent and nonpermanent Armed Forces hospitals, adoption of the principle of built-in clinics rather than the conventional outpatient department, adoption of the back-pressure, arm-lift method of artificial respiration, coordination of research in the medical and health fields with the Research and Development Board, the Armed Forces blood donor program, point credits for reserve officers attending the section on military medicine of the American Medical Association, and many others.

These accomplishments are sufficient to indicate the great value of the important step that was taken when joint action of the Armed Forces medical departments was obtained through the Armed Forces Medical Policy Council.

MORRIS FISHBEIN

*Obtainable through the Government Printing Office, Washington, D. C., $5.25 per year.

Men of Medicine

Bones and Battles

Dr. Paul Budd Magnuson, called recently by President Harry Truman to head a "commission to study the nation's health needs," is especially fond of recalling one encounter during his last visit to England.

The meeting, in a town of about 10,000 population, was with the Labor M.P. Dr. Magnuson had noted that, in this small community alone, three government bureaus were being manned to run the local end of the government.

"I have one question, Mr. M.P.," he recalls telling the Laborite. "What percentage of the money collected from the people actually goes to the support of a bureaucracy rather than into the actual giving of good medical care?"

Dr. Magnuson's guess was that about 40 per cent of the money was actually finding its way into health care, and to a pretty poor form of care at that. The M.P. said they had no figures on this phase of the program, to which Dr. Magnuson replied, "Probably you won't release them if you ever get them." He adds now, in recalling the episode, "If I were betting, I'd wager most of the money goes to support the six-copy brigade. Long live red tape and bureaucracy!"

The Magnuson fondness for this incident dramatizes the fact that, whatever may be thought of his own unspoken motives, Mr. Truman chose no soft rubber stamp or advocate of compulsory health panaceas when he asked Paul Budd Magnuson to head his new health commission.

A distinguished orthopedic surgeon, and one of the men chiefly responsible for having transformed VA medicine from a national scandal into a thing of promising professional beauty, Paul Magnuson is a blunt, outspoken fellow whose medical ingenuity is exceeded only by his zeal for direct action.

"I'm simple-minded, so I have to think simply," he says, with characteristic good-humored terseness. He knows, as he's wont to observe, that "A lot of people want miraculous green pills with fancy fringes" to cure them of their complaints "without too much effort on their part." But he will not permit himself to be an ostrich-like apologist for social shortcomings he believes need attention.

When General Omar Bradley and Major General Paul R. Hawley summoned him from Chicago to Washington in 1945, to work with the VA, Dr. Magnuson made his position clear.

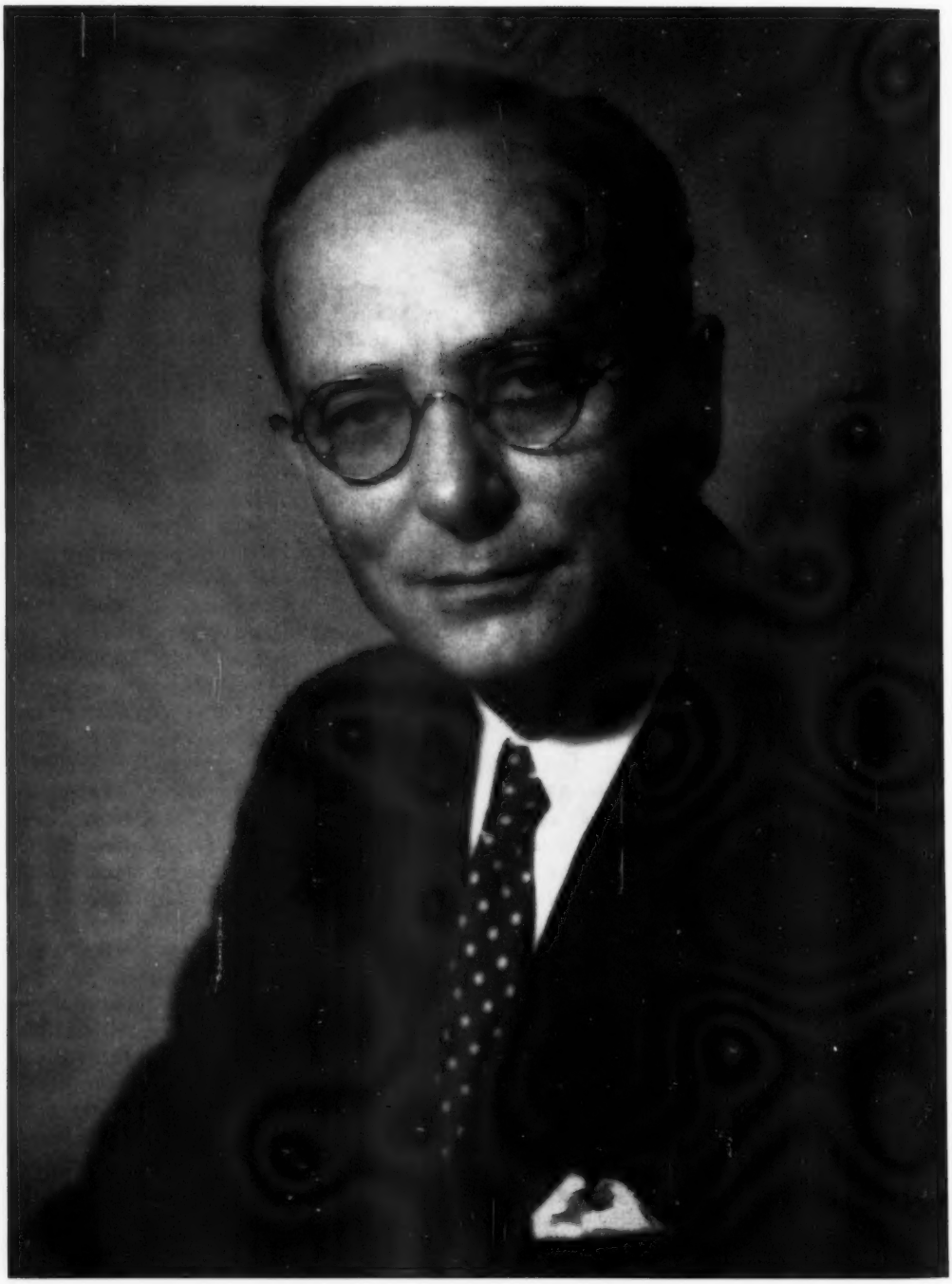
"One thing I hope to do," he announced, "is to clean out those skunks and chiselers in the medical profession who have been running a racket. They are few but they give the public a bad impression and spread the smell to the profession at large."

The Illinois State Medical Society pleaded with Dr. Magnuson to be "less intemperate." But the *Journal of the A. M. A.* agreed with the Magnuson position, observed "A house cleaning has been too long delayed." Dr. Magnuson answered on his own.

"You and I know this charge is true," he told his Illinois colleagues. "Let's face the facts, and get rid of the few people who bring disrepute to the profession." With the evident backing of Bradley and Hawley he also took wry note of the traditional VA tendency to pursue a construction policy not in keeping with the available supply of medical and nursing personnel: "As far as care of veterans is concerned, hospitals without doctors are useless—little more than piles of bricks.

"They become meaningful only when they serve to bring sick people together with good doctors and their co-workers.

"A hospital is just a factory. The sick patient



PAUL B. MAGNUSON, M.D.

is your raw material, doctors, nurses, technicians and others are your labor, and x-ray machines and other devices your equipment. The sick person made well is your product. Its quality depends on the brains of the people doing the work.

"I am looking to the day when every VA institution will be a teaching hospital, operated along university standards, and the quality of care will be such that every medical school will want its best men working in them."

It was a daring concept to broach, in the shadow of a well entrenched, biliously bureaucratic VA system which until then had not even arranged for intern work in its hospitals.

But Paul Magnuson had the topside brass foursquare behind him. In less than five years, the one-and-a-half-page reorganization plan he had brought with him to Washington was in widespread operation. As he says, "Some 60 medical schools and their Deans' Committees were cooperating, mostly on a nonpay basis, to make VA medicine fulfill its promise. Bright, young men were at work in VA residencies. Many VA oldtimers, freed at last of the crippling disease of days spent filling out forms, were getting back to being doctors."

After Bradley and Hawley had left the VA and bureaucracy showed signs, last year, of moving back in, Dr. Magnuson's dismissal was the trumpet that sounded an alarm to the nation.

"There was nothing personal in it," he says. "I simply wanted to focus attention on the problem. I knew—and said so—that if we weren't very damn careful, we'd see a return to political control and inferior medical care."

The gambit apparently worked. Last July, a senate subcommittee, obviously mindful of the professional organizations which had rallied to Dr. Magnuson's support, wrote a report backing him to the hilt of his scalpel.

In fact, the committee even recommended legislation requiring that the power over VA hospital operation, which Magnuson and Hawley had enjoyed as medical directors, should be left untouched, lest "bureaucratic niche-carvers forget that the sole purpose of hospital operation is the care and treatment of patients."

Such a law has not been passed, but Dr. Magnuson currently is satisfied that the administrative threat to sound VA medical care has been at least temporarily dispelled.

A lesser man might have quailed or compromised in this battle with bureaucracy. But this was not the first experience of its kind for the mighty Swede, Magnuson. In the second decade of the twentieth century, he had his initial tangle with civil service, as first medical director of the Illinois Industrial Commission.

Recalls Dr. Magnuson: "It was the greatest clinic imaginable, working at a time when there were practically no compensation laws and the general industrial rule in case of injury was 'Settle for a dollar while the blood flows.'

"But we had to operate with a bunch of dumb dodos, civil service clerks and assistants, some of whom couldn't even write or punctuate. It was a constant fight against incompetency."

Dr. Magnuson must have met it head-on, with the greatest possible directness. An inventory of his career suggests that this impatient but revered, gray-haired stalwart, whose eyes twinkle behind steel-rimmed glasses, has been attacking professional problems with the same directness.

He was still a medical student at the University of Pennsylvania, when, in 1908, he reported on the first successful research in surgical technic for the lengthening of shortened leg bones.

As early as 1916, he was forecasting the belief, just now coming into vogue, that low back pain and sciatic pain were to be attributed more to strain of the ligaments of the low back than to any other one cause. Instinctively alert to the drawbacks of overspecialization, he refrained from taking his orthopedic board examinations until he was 60, although he is a founder member of the American Board of Surgery.

Yet, when he went to the VA in 1945, it was on leave from distinguished performance for 15 years as professor of surgery and chairman of the department of bone and joint surgery at Northwestern University, and as author of a medical classic, "Fractures," currently in its fifth edition.

Now 67, Dr. Magnuson was born in St. Paul, Minnesota, the second child (and second son)

of Charles Augustus and Melinda Graham Magnuson. Dr. Magnuson remembers his father, president of a Minneapolis grain elevator company, as having been "the best-educated man I ever knew." He had a prodigious memory, which Dr. Magnuson still envies, and could discuss astronomy, art and literature with staggering facility.

Fondly, Dr. Magnuson recalls his mother's favorite appraisal of her son: "You're always positive, and sometimes right."

It appears to have been a remarkably healthy family. Until the senior Magnuson died in 1945, at the age of 89, there never had been a serious illness or any major surgery in the clan.

Curiously, the death of Dr. Magnuson's father was soon followed by the demise not only of his mother but of his sister, Louise, a Vassar graduate who used to spend girlhood days trying to drum school lessons into brother Paul's noggin.

In such a sturdy family group, it is not unreasonable to wonder how Dr. Magnuson came by his early devotion to the practice of medicine. The fact is, he was only a lad of seven when, thanks to one of the rare episodes of malaise in the family, the die was cast. A few days after the birth of his baby sister, Ruth, their mother developed a breast abscess. While the family doctor drained it, Paul held the basin. "From that day on, I guess I was a goner," he grins.

For his premedical work, he attended the University of Minnesota—four years in which, he confesses, laboratory puttering and summer jobs delivering groceries, selling advertising, and working with a railroad survey crew in the state of Washington claimed more of his attention than book larnin'.

The father of his roommate at the University of Pennsylvania medical school had to struggle about with a short leg and crutches, the result of a polio onslaught. This circumstance, Dr. Magnuson believes, was what drew his attention to matters of bone and joint surgery.

In the University of Pennsylvania laboratories, young Magnuson set to work studying operative technics for lengthening shortened leg bones. In the course of this project, he became more intimately familiar with the general undesirability and unreliability of steel screws and plates for holding fractures.

Out of this medical school investigation came the memorable experiments which made Dr. Magnuson the first to use ivory plates and screws as fracture-retention apparatus. He became also the first to use a mechanic's-type tap for cutting a screw thread in bone fragments.

By haunting the Baldwin locomotive works, across the Schuylkill River, he found the mechanical inspiration for this work.

"I was fascinated to see the way they handled the drive shafts, the methods they employed to hold together the drive shaft and the hub of the wheel," he says, still enthusiastic after all these years—and swiftly pencilling a drawing on a scratch pad to illustrate his point.

"After all, what are bones but shafts?" He took the lesson of the locomotive back to school with him, successfully applied it in developing his technic for plating transverse fractures.

Because existing chisels were unsatisfactory, couldn't be counted on to split a bone straight, student Magnuson developed a motor-driven bone saw, which he perfected the year of his graduation, to use in cutting bones for splicing and lengthening.

Except that, these days, universal current motors have supplanted the primitive power unit Dr. Magnuson first employed, the Magnuson bone saw remains in general use largely as he invented it.

In early practice, he used to have to make certain, in advance, of the type of electrical current likely to be available on a case. Unexpectedly rushed in to treat the son of the town banker in Fulton, Missouri, Dr. Magnuson found himself with an A.C. motor in a D.C. town.

"A mechanically gifted inmate of the nearest state hospital for mental diseases rigged up a new motor for my cable. I not only used it to power my saw for the case in Fulton, but used it for 20 years after," he says.

THE zeal for tackling inviting research problems had its inevitable effect on the more orthodox phases of Magnuson's medical school career. Not until many years later, the bone surgeon says, did his father learn that the extra

allowances about which he'd often wondered had gone into research, not revelry.

Where did Dr. Magnuson stand academically in his class at medical school? The mischievous Swede confesses heartily, "Hell's bells, I was in the bottom 10 per cent! I was just too busy trying to do the things I was interested in. Consequently, I got a lopsided education which has always been a handicap."

With his own peculiar brand of modesty, he ascribes many of his mechanosurgical developments to his chronic ambition to save himself work.

As an intern under Dr. Edward Martin at the University of Pennsylvania, Magnuson found himself—as the huskiest fellow in sight—always assigned to hanging on to somebody's fractured leg. In what was, for its time, a novel traction device, Dr. Magnuson rigged up a pedestal with a puiley through which he passed a rope, one end tied with a clove hitch to the patient's ankle, the other end secured to a parcel of window weights.

"I could see no reason for my working as long as the law of gravity was," Magnuson says, twinkling.

Early in 1910, he was called to Chicago to serve as assistant to the world-famous bone surgeon, Dr. John B. Murphy. Later, he became associated with another leader in the profession, Dr. William E. Schroeder.

Dr. Magnuson's mother saw to it that such recognition didn't go to her boy's head. "Son," she liked to remind him, "I feel sorry for your patients. You never could drive a nail in a plank without splitting it."

Added to the staff of Chicago's Wesley Memorial Hospital in 1912, he performed the first leg-lengthening operation there two years later and became embroiled in a controversy which ended in the complete justification of his technic.

His first office was located in two rooms over a saloon, hard by the entrance to the Union stockyards on Chicago's teeming southwest side. There was an extension phone in the saloon, so the bartender could take calls during the doctor's absence, double in brass as the office attendant Dr. Magnuson couldn't yet afford.

In addition to his post as first medical director of the state industrial commission, Dr. Magnuson found time for other early associations as chief surgeon of two railroads (Chicago Junction and the Chicago and Alton) and as medical consultant to a number of coal mining and other industrial firms.

His teaching career at Northwestern University began in 1913 and in subsequent years brought him a startling measure of devotion from students who found, in his dry humor and blunt imagery, an ideal kind of open sesame textbooks couldn't provide.

Says one former resident admiringly, "I learned more about fractures from making rounds with Dr. Magnuson than I learned at any other time or place in my training. The man has an eagle eye and split vision; he can be in hearty conversation with a patient and still be spotting some obscure maladjustment in the weights and pulleys."

Seeing service in World War I as a medical corps major, Dr. Magnuson tried to keep his Chicago office going, in absentia, by leaving it in the hands of an assistant, finally gave it up. "Well," he philosophized at the time, "I can't stay at the stockyards all my life."

The bartender, they say, was inconsolable.

RETURNING to his Chicago practice in the years between two great wars, Dr. Magnuson found himself drawn to the problem of arthritis; his work resulted in the origination of surgical treatment for several types of arthritis. Notable is his operative method for treatment of degenerative arthritis, on which he reported in 1941 in *Surgery, Gynecology and Obstetrics* with what he believes to have been one of the first medical papers employing color-photo illustration.

The work was done at Chicago's Passavant Memorial Hospital, where Dr. Magnuson has been a staff member since the institution's opening 23 years ago.

Also to Dr. Magnuson's credit are the surgical procedures for repair of un-united hip fractures and for correction of recurrent dislocation of the shoulder.

In the mid-twenties, Dr. Magnuson himself developed a painful back condition. Experi-

Dr. Magnuson as a medical student.

menting with various positions, he discovered he could get maximum comfort from sitting on a high stool with his legs curled back around the stool.

From this perch, he dissected scores of cadavers, learned enough to strengthen the premise he'd stated first in 1916: Low back pain and sciatic pain are to be attributed more to strain of the low back ligaments than to any other cause, and operation for removal of intervertebral disks, without definite evidence of root pressure, is not justifiable until every other treatment method has been reasonably tried.

In pursuance of this premise, Dr. Magnuson became one of the first U. S. orthopedists to use traction as a therapy in back cases.

THE story of Dr. Magnuson's entry into the field of VA medicine is professional history of the most solid sort.

It was in 1942, anticipating immense new demands for a dynamic brand of veterans' health care, that Magnuson first presented to General Hines, then VA administrator, a plan for associating veterans' hospitals with the medical schools, to become in time teaching institutions. At that point, Dr. Magnuson recalls, the VA institutions were not even qualified for the use of interns.

His plan met with a cold reception. There the matter rested, although it had no effect, of course, on the extent of war work done by Dr. Magnuson; he organized and supervised the course at Northwestern University for selected army medical officers and served on the National Research Council, the committee on orthopedic surgery and as a civilian consultant to the surgeon general.

In the fall of 1945, Dr. Magnuson was visited in Chicago by Frank Stinchfield, a brilliant fellow who had been successively, under Magnuson, intern, resident and assistant, had entered army service as a first lieutenant, achieved distinction as the youngest eagle-colonel in the medical corps.

Asked Stinchfield (now, at 40, a professor of surgery at Columbia University medical school): Would Dr. Magnuson advise him whether or not to accept an invitation to assist General Hawley in establishing a clean deal in VA medicine?

"It depends on whether you want to be a surgeon or an administrator," was the Magnuson response. Stinchfield went off satisfied that he should decline the offer; before many days had elapsed he was back on Dr. Magnuson's North Michigan Boulevard office doorstep. Would Dr. Magnuson himself accept the VA bid?

Dr. Magnuson grins upon recalling his answer. "Hell no!" he said. "I've got between 60 and 90 cases in the two hospitals, I'm working 16 hours a day, six days a week; how can I consider a thing like that?"

Next day, General Bradley and General Hawley came to Chicago to see if the Magnuson word was final.

"All I know is how to practice medicine," Dr. Magnuson says he told Bradley and Hawley. "I'll probably end up getting you both in jail."

Countered General Bradley quietly, "We'll expect you to try to keep us out, but if you have

Dr. Paul B. Magnuson, General Omar Bradley and General Paul R. Hawley.

to go to jail in order to get the job done, we'll go with you."

Says Dr. Magnuson: "As soon as I met General Bradley, I knew I was with an honest tower of strength who would give me support from the top." Paul Magnuson agreed to serve in Washington for six months, as "acting assistant medical director for research and education." When he left Chicago for the capital, his one and-a-half-page master plan went with him in his briefcase.

Action came quickly. Within six weeks, in response to telephone barrages from Dr. Magnuson, Northwestern University and University of Illinois medical schools jointly had placed 53 residents on duty in the VA's mammoth Hines General Hospital, in Chicago.

Three weeks later, thanks to cooperation from his old friend, Dean Harold Diehl of the University of Minnesota medical school, Dr. Magnuson had 26 residents at work in the VA hospital in Minneapolis.

"We went through like Grant took Richmond," Dr. Magnuson recalls. With the team operating on the rule-of-thumb principle that only the truly ill merited admittance to VA hospitals, Hines, for example, was emptied of 40 per cent of its patient load, and surgical bed turnover was hiked 270 per cent.

With the solid backing of Bradley and Hawley to put the punch in his promise, Dr. Magnuson was able to telephone other professional friends throughout the U. S. and tell them, as he was to tell VA doctors: "These are the standards. We want these hospitals to be run just like university hospitals. If anybody gets in your way, they'll have Bradley and Hawley to answer to."

Magnuson was the architect for the widely praised plans under which the VA provides attractive-paid specialty training courses to qualified residents, draws into its research and treatment activities the skill of leading consultants.

He said, at one point during his VA tenure: "I hope no doctor stays with VA more than 10 years. We want a continual inpouring of fresh, young men."

Washington soon learned that he was there. One day he telephoned a man named McNamara in the Budget Bureau.

"I heard you're the toughest Irishman in the government," Dr. Magnuson said. "Well, now

you're up against the toughest Swede. I wish you'd either send a fellow over to give my lecture on bone and joint surgery while I make out my budget, or send somebody here to translate my ideas into dollars and cents."

In September 1947, Dr. Magnuson became acting assistant medical director for professional service; the following January, upon the departure of General Hawley, Dr. Magnuson was named by Carl R. Gray (the newly appointed administrator) to be the agency's Chief Medical Director.

Dr. Magnuson was operating on the premise that 120,000 beds represented the maximum which could be serviced with good medical care on the basis of available professional personnel.

In the fall of 1950, Dr. Magnuson voiced a warning against "hospitalitis," predicted that without vigilance VA institutions might become mere "country clubs."

When he finally departed from the VA early last year, colleagues were quick to rally 'round. Dr. Kellogg Speed, as president of the American Orthopedic Association, viewed his leaving the VA with "dismay and alarm." Protests came also from the Association of American Medical Schools and the American College of Surgeons.

By way of demonstrating his continued concern for an important cause, Dr. Magnuson—when he and the VA parted company—left his long-time secretary, Lola Winters, on temporary duty to assist his successor, Vice Admiral Joel T. Boone, in getting oriented. This was no little sacrifice.

Miss Winters, soft-voiced and gray-haired, went to work for Dr. Magnuson as a young girl, shortly after his return from World War I duty. She tenders him a loyalty that is paralleled by patients like the little Italian girl upon whom Dr. Magnuson operated as a clinic case 25 years ago, and who has sent him thanks every Christmas since then.

"I swear I was still in my army uniform when Miss Winters and I first met, but she doesn't remember it," Dr. Magnuson sighs. "It isn't very flattering."

At the time, he had returned to battling incompetency in the civil service. "I needed a girl with brains," he says. He, himself, was netting only $200 a month, but upon spotting Miss Winters holding down a state job, he lured her into his employ with a salary boost of $50.

"The day she came to work, I had a mess of trouble with my four-cylinder Hupmobile," he says. "My language in those days would have made a river pilot pale and I was using it."

Miss Winters went home that evening and told her mother, "Never in my life will I be able to stay around that man."

Subsequently, the sight of Dr. Magnuson tenderly taking extra time with patients—making little sketches of their ailments, never holding out false, grandiose hopes for the results of impending surgery—persuaded Miss Winters to stay on.

Among the hazards Miss Winters has successfully weathered through the years has been Dr. Magnuson's chronic failing when it comes to grammar. Early in his VA service, while it still appeared that he might need to stay only six months and Miss Winters was in Chicago tending to routine office matters, Dr. Magnuson rapidly went through four or five secretaries. He couldn't understand why the girls were coming up with such gravel-phrased versions of his dictated correspondence.

Another Winters effort of long standing has been her protracted effort to educate telephoning patients on the preferred pronunciation of the doctor's name.

"Among the Swedes of Minnesota, it was Magnu-SON," the doctor grins. "I had to come south to hear it pronounced MAGnuson." The preferred version—also the one most commonly used among Chicagoans—is MagNUson.

The doctor was married in 1910 to Alice L. Hasson, his high school sweetheart from St. Paul. Dr. Magnuson says it was his decision for them not to marry until after the termination of his medical school and intern periods. "It meant an eight year engagement, but I didn't want to marry without being able to support a wife," he says.

The couple had one son, Paul, Jr., now a Chicago advertising man and father of three youngsters, one of whom—Alicia—can wrap Grandpa Magnuson around her little finger.

The first Mrs. Magnuson died at 47, following 12 years of invalidism. In 1931, Dr. Magnuson and his present wife, the former Laura Thompson of Chicago, were married in an old meeting house on the pike near Dundee, 40 miles northwest of Chicago.

It was in the vicinity of Dundee where Dr. Magnuson, ever a lover of horses and dogs, had bought a 50 acre farm in 1927, to recapture some of the childhood pleasures he'd enjoyed during visits to an uncle's Minnesota farm.

With the present Mrs. Magnuson enthusiastically pitching in, "Pond Gate Farm" became a widely-talked-of Chicago area institution. By the time the Magnusons went to Washington in 1945, it had grown to 600 acres.

In 1933, recording the early-springtime hegira of the Magnusons from their city apartment, a Chicago society reporter observed, "The Magnusons are always the first to move out to the country and the last back in."

The doctor was no gentleman farmer. Until the VA call, it was his custom to drive the 80 miles to and from Chicago four days a week, to attend to office, hospital and medical school duties; from Friday to Tuesday of each week, he played the active farmer—operating a combine, inspecting the dairy herd or range cattle, getting hogs off to market, actively pitching in on the cultivation of alfalfa, corn and oats crops. Mrs. Magnuson managed the herd of Holsteins.

Beginning about 17 years ago, Dr. Magnuson operated on part of his land a game-raising and controlled-shooting preserve where he supervised the annual raising of about 7000 ducks and 5000 pheasants, also planted many feed patches.

"The state game laws weren't exactly suitable to the raising of game in connection with a shooting preserve, so I got the laws changed and modernized." He grins, adds embarrassedly, "Fact is, I helped write the new law." It made legal the raising and releasing of birds where no more than 75 per cent of those tagged by the raiser are shot.

The Magnusons trained and rode polo ponies and hunters, and used to make at least one annual excursion to a remote Mississippi log cabin retreat, where they found quail, duck and pheasant hunting "a welcome change from city parties." Dr. Magnuson, gallantly deferring to his vivacious wife as the better trout fisherman, says the trout streams of the Michigan peninsula, Minnesota and Canada still hold a mighty appeal.

"But no more partridge hunting," he sighs. "My days of climbing over windfalls are through. As for polo, I quit it when I was 50, after breaking both my wrists."

PERHAPS nowhere is the Magnuson fetish for simplicity and directness better illustrated than in the current edition of his volume on "Fractures."

One of its new chapters is on the "farmyard" reduction of fractures.

Says he, in discussing its message, "I can reduce any fracture of the long bones with some bandage, a sheet, two pulleys and a piece of clothesline."

His own first work was in the development of ivory screws and plates for fracture retention, yet he says candidly (in collaboration with his partner, Dr. James K. Stack) in the preface to the fifth edition of his book:

> "The use of gadgets in treatment of fractures has seemed, during the past few years, to overshadow the common sense and simple approach to the problem.
>
> "While we do not deprecate the value of many of these mechanical appliances . . . it is our belief after an extensive experience . . . that only one splint is fundamental—the Thomas splint.
>
> "If the principles of its application and use are understood, almost any mechanically minded young doctor, if he has a basic understanding of anatomy, physiology and pathology, can figure out how to reduce almost any fracture and the type of retention apparatus necessary to hold it in position. Pins and screws and wires may be useful in certain selected cases, but certainly not to the extent to which they have been used by enthusiastic operative surgeons in the last ten or fifteen years.
>
> "It was clearly demonstrated, during World War II, that suspension traction is still the method of preference; this was no surprise to those who had been using it and to those to whom it was taught thoroughly in student days. Suspension traction may require more personal supervision by the surgeon than other methods, because it has to be watched closely to see that it remains true suspension and traction until

union occurs. The same muscle exercises and joint movements can be carried out in most instances with suspension traction as with pins and wire.

"During the war, Navy surgeons advocated the use of pins in a great many cases; this was justifiable under the circumstances in which many of their patients were treated. Certainly we would all choose for ourselves or our patients a form of treatment which would permit the patient to get out of bed in an emergency and, if necessary, keep himself afloat, in preference to being encumbered with a heavy cast or suspension traction apparatus. But in a civilian hospital where such emergencies do not occur, the making of a tunnel through bone, which communicates with the skin and outside, lays the patient open to an added risk. Certainly it is easier for the surgeon to put in pins or put on a plate to fix a fracture, but it is not necessarily the best thing for the patient. As a matter of fact, we feel it is quite unnecessary in the great majority of cases if due consideration is given to all the factors involved in the specific case."

Also a part of the "Fractures" volume is the First Edition preface, in which Dr. Magnuson exhibited his readiness to examine the next man's favorite hobbyhorse, but foreswear it until pragmatically tested.

"All the methods described in this book have been thoroughly tried in practice," he wrote. "There are many more which are as good, there may be some that are better, but these have worked!

"And with thought and attention to detail they will work in the hands of any man, because they are simple and because they all take into consideration the anatomy and physiology of the parts under treatment, with the mechanical features simplified so that they may be applied without any great amount of special equipment."

To suit his Washington sojourn while with the VA, Dr. Magnuson obtained for himself and Mrs. Magnuson a three-story home on O Street in Georgetown, eventually took on some teaching stints at George Washington University.

Following his exit from the VA, he began shuttling between Washington and Chicago, unable to resist the temptation to get back to active surgery, especially reconstructive work on joints. He was beginning to believe he might get around to completing his book on backs, for which he wrote some 1500 pages in 1932.

"There it lies," he told a Chicago office visitor recently. "Will I finish it?"

A few days later he was invited back to Washington; in due course, there came the White House announcement that Dr. Paul B. Magnuson had been given a free hand to select a commission, assay the nation's health needs at presidential request.

In 1949, Dr. Magnuson—quietly seeking a compromise between the extreme positions figuring in the compulsory insurance squabble—proposed his own plan. It suggested establishment of a chain of diagnostic clinics, to be staffed by medical school, public health and VA personnel.

Examinations would be on a free or part pay basis, treatment at regular rates, with government making up the difference for those unable to pay full fare.

"Almost any doctor can treat 90 per cent of the known ills, if he has the diagnosis," Dr. Magnuson says. "It's when patients are being hustled about from one specialist to another for diagnosis that expense mounts."

No doubt, this will be one of the patterns due for examination by the new commission. Those who plan to keep their sights critically trained on the group may find interest in what some Magnuson friends think is a meaningful recollection of his pheasant-raising days.

It was the year the pheasant hens got peevish.

"If you're going to deal with hens, you must be patient," Dr. Magnuson said later. "When the pheasants lost their patience, I lost mine and we had a few for dinner."

Robert H. Schulman

A Surgeon's Viewpoint*

RICHARD B. CATTELL

LAHEY CLINIC, BOSTON

It gives me great pleasure to have the privilege of presiding over this group this evening. We appreciate, Dr. Casberg, your welcome and the welcome on the part of the profession of St. Louis and environs. I would like to take this opportunity to express our regrets, however, at the inability of Dr. Sante, because of ill health, to be here.

I should like first to welcome the ladies to this audience and to assure them that the purpose of this gathering this evening is not for postgraduate instruction but rather for a congenial get-together in one of the few times at the meeting in which they may see their husbands.

This is the thirty-sixth Assembly of this Association and there has been a great change over this period, beginning in 1916 and continuing to this time, with but one year in which an Assembly was not held because of unavoidable reasons.

As president of this Association it also gives me the opportunity to call the attention of the ladies to the type of Assembly this is, for we have a medical meeting at this time that is unique and different from all other medical meetings with which I have been associated or which I have attended. We have, in the annual session of this Assembly, perhaps 60 or more speakers selected because of special attainments and special abilities to teach and to present material before audiences of this type. The subjects presented cover all fields of medicine, whereas the usual medical meeting is divided by specialties, divided by interests, and frequently less well attended because of that. Here we may come as surgeons—I for one being a surgeon—and listen to those who are specialists in other fields and can thus keep abreast of progress in other branches of medicine. We come to a meeting such as this to broaden our interest and knowledge, which we cannot do by attending surgical meetings alone.

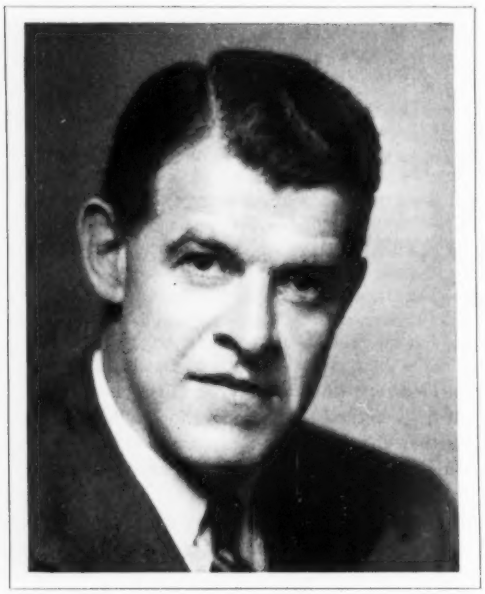
*Presidential Address presented at the 1951 (St. Louis) Assembly of the Interstate Postgraduate Medical Association.

This type of Assembly permits the presentation of new methods of therapy and disciplines as soon as they can be established; it permits a review of older subjects, all in a practical manner, so the information can quickly be applied in our own practices. As a matter of fact, the distinguished physician seated on my left, your president-elect, Dr. Cyrus Sturgis, in presenting a clinic today was so clear in his elucidation of his subject that I felt I should, as a surgeon, be competent in the treatment of pernicious anemia.

This is a unique form of postgraduate education and it deserves real commendation. Those sitting in the front of this audience, the officers and trustees, deserve a great deal of credit for their part in developing this particular Assembly, which I would like to acknowledge publicly at this time. This type of postgraduate education is very important, and I think many of us, interested in postgraduate education, appreciate the fact that today the United States stands pre-eminent in this field. There is a constant stream of physicians from all countries of the world who not only are coming, but more wish to come, for the opportunities of postgraduate training which are present here and not present elsewhere.

The character of this meeting, recognizing it for what it is, must be preserved in a continuation of this postgraduate type of training. Unless it does persist in a fashion such as has been developed in the past, it cannot continue to serve the useful function which it is performing today.

This brings us to another consideration of the training of a physician, that is, the graduate training for the period immediately after finishing medical school. This education must be conducted in hospitals. While a minimum requirement to practice in some states is one year, most physicians will prefer the additional experience to be gained from several years of residency or fellowships. I should



RICHARD B. CATTELL

like to mention two considerations relative to this period of training: The first has to do with the problems of the hospital furnishing this training and the second is concerned with the individual seeking this training.

Many of the hospitals in America—in your own communities—are faced with the difficult problem of obtaining interns and residents to carry on the essential functions of medical care which they are attempting to provide. This is not true of the university and teaching hospitals where the positions are readily filled. The general hospital will need help in meeting this problem and it may receive aid to some degree from the teaching hospital.

There is a possibility, however, which I would like to call to your attention whereby this number of places might be increased and whereby further candidates can be trained in a program that will not disrupt the programs now in progress in our universities and large general hospitals. This can be done by means of affiliations of the smaller general hospitals with the university centers which are represented by Dr. Sturgis and others in the teaching field. By this means the number of interns and residents, but particularly residents, who are assigned to a university service or other large hospital service can be assigned to other hospitals which will meet certain standards so as to be of advantage to a man well along in his training, and to contribute to that training through the clinical facilities which are present in that hospital. Unless this is done we will find that our smaller general hospitals will not be properly staffed and manned. This is an opportunity that the universities and other general hospital services can offer to the smaller general hospitals in smaller communities.

The third problem that I should like to discuss very briefly relates to our medical schools. We have 79 approved schools in this country and at present there are approximately 26,000 students enrolled in these schools. This is a 4 per cent increase over last year, but even more over the past few years, for in the past 10 years there has been an increase of students in our medical schools of 25 per cent.

We hear a lot of propaganda relative to the great need for increased numbers of physicians in this country. We believe that this will be answered properly by this increase in enrollment in our medical schools and also by an increase in the number of medical schools themselves. Without much question, there will be 10 additional schools that can be approved and have a standard similar to those already established within a short period of time, but this cannot be done overnight because one cannot assemble the teaching group required for an additional 10 medical schools without training them over a period of years.

Actually the number of doctors in this country today, as you probably know, is over 200,000. The last figure given by the American Medical Association is 209,000, but this is not the number of practicing physicians—merely the number registered. Not only has the number of physicians in this country increased in recent years beyond the increase in population, but also we as physicians, because of the new disciplines which have been developed and the new treatments of diseases such as antibiotics and other drugs, have become 30 per cent more proficient in the work which we accomplish. We must not be panicked by figures promulgated by parties disinterested primarily in medical practice into the feeling that medical schools must expand themselves beyond the possibility of their carrying on satisfactory education. Without too great dilution in the teaching in our schools, an increase of enrollment of 10 per cent has been effected in many schools and can be in additional ones.

We as practicing physicians would like to ex-

press our sympathy for the problems of the medical schools, particularly as to the cost of carrying on medical education in our medical schools. The figures available for the past year show that there has been more than a 15 million dollar deficit in medical education that must be met by some means. American medicine has already risen, as you know, to help meet this emergency by the creation by the American Medical Association of a fund to further medical education and to support medical education in the amount of a half million dollars to begin with, and a national committee of interested citizens has been created to help raise a fund to aid in this matter.

Actually, as has been proposed, if each of the practicing physicians would give $100 to his own medical school, or to this fund, this deficit would be wiped out. Obviously this would be the best way to continue our system of private enterprise if sufficient interest could be aroused. I would like to encourage it in a somewhat different and modified way which offers some prospect of success, because I believe that this can be done only on the local level. I think it would be possible for alumni associations of our various medical schools to get support of this sort paid as an annual contribution rather than to seek capital sums for endowment. This has already been done in a number of our other medical schools. I do not think it is possible for one to approach the profession of this country as a whole to contribute a fund of this magnitude—of 15 million dollars—but I do believe that there is a possibility on the local level.

Relative to additional support to the medical school, various functions are at present carried on by medical schools, most of which are important, that must have separate cost accounting. We should know the cost of research, teaching of students, postgraduate education and of other functions. It will be necessary to obtain funds from different sources for certain of these activities.

Whatever the source of these additional funds, they must not influence or change the present direction of our medical schools.

The next point that I would like to mention to you briefly has to do with the hospital situation in the country. Hospital construction, as you know when you survey your own communities, has gone ahead by leaps and bounds in recent years; it has had federal aid at a level at which perhaps federal aid is necessary. Since the Hill-Burton Bill has been in effect, over 1600 projects for hospital building have been approved and, of these, approximately 1500 have already been finished and are in operation or are actually under construction. This has resulted in the building of 600 general hospitals, and the important reason why we as a profession probably must accept federal aid in this regard is that 60 per cent of these hospitals have been built in towns of less than 5,000 population, whereas only 7 per cent have been built in cities of 50,000 or more.

The actual increase in beds available for medical care in this country has surpassed the increase in population. To be sure, it will be necessary even where the population is not increasing in certain communities to increase the hospital construction more than would be proportionate to the population, but that is a local problem.

The building of community hospitals, which has been furthered and sponsored by the American Medical Association, will help to answer another problem with which many of you sitting in this room are quite concerned, and that is the ability to make hospital beds available to physicians in their communities. There will always be some hospitals with specialized care to offer, specialty hospitals where beds cannot be made available to general practitioners in the area, but the community hospital should have as one of its primary functions the ability to make the care of patients available to the physicians who have their responsibility.

All of us should be encouraged by what has been accomplished in the last two or three years relative to the protection of private enterprise as concerned with the practice of medicine. Most of us are familiar with the fact that increasing numbers of patients are enrolled in a voluntary plan of health insurance. In 1950 over 77,000,000 people were protected against hospital expense by this voluntary plan of health insurance, which I think most of us will accept as the best means of handling the problem in this country today. Medical expense for professional services is less well covered but in this regard progress is being made, for during 1950 there were 55,500,000 people protected against major surgical expense and 21,500,000 protected against medical illness as well.

That this plan of voluntary insurance and protection for our population and our citizens offers a real possibility of answering the major problems relative to the costs of medical care should be fairly well proved by the rapid increase in people becoming enrolled under such plans. There has been an increase within one year of 17 per cent

in hospital protection; there has been an increase of 32 per cent in surgical protection, and 28 per cent in medical protection. The latter, while it is important, will undoubtedly lag because it does not involve as great a risk to the individual as do the other illnesses with which we have to concern ourselves.

The next problem that I would like to call your attention to has to do with an acute problem in this country today, and that is adequate nursing care that can be made available for hospitals and outside of hospitals as well. The best of our minds relative to hospital care and administration and medical care have been focused upon this problem and many advances have been made to improve the situation. Among these has been the lightening of the load of nurses by those less well trained and without a full nurse's training. For example, one of the biggest helps has been the use of the untrained ward aid in carrying out the menial duties in the hospital wards. Similarly, in certain states, particularly, and in my own state of Massachusetts, an important development has been the use of attendants who can carry on most of the nursing disciplines outside of unusual medication, providing they are done under proper direction.

One of the serious obstacles to providing adequate nursing care at present has been the fact that nursing education with its long period of training has been so expensive and so protracted that many girls who would like to go into nursing have been unable to do so. Furthermore, the standards for an ordinary course of nursing have been such that many girls, having finished a high school education, are not able because of lack of certain sciences or other requirements to take up nurses' training. I think this is a great mistake. To be sure, it would be advantageous for those girls entering nursing, who will go on to a teaching position or to a specialty job of nursing, to have additional training even beyond a high school education and go on to college before entering nurses' training, but that is not a need for the major portion of our nurses who are to carry on the work of our hospitals and take care of the ill. Furthermore, it seems to me that the nurses' training course can be decreased in time without interfering with the proper carrying out subsequently of the nurses' duties.

More and more training schools are affiliating themselves with colleges or universities in order to be able to offer a college degree. This will result in fewer nurses available for the bedside care of the patient as these women so trained will be more interested in teaching and administrative positions and will not fill the major need of the actual care of the sick.

There are approximately 61,000 girls entering nurses' training this year. Probably an additional 10,000 are needed. It behooves many of us as physicians and physicians' wives to encourage an additional number of nurses to enter this most engaging and attractive field, and I think that if we will do our part in attempting to increase this number it will be advantageous.

The nurses who wish to go on and do additional work of a specialized nature probably constitute less than 10 per cent of those finishing nurses' training. There is every reason for such as these to go on with additional training in order to qualify for teaching positions, running training schools, surgical nurses, public health work, and so forth.

There is one situation which I am afraid we have no answer for relative to the nursing shortage. There is a definite competition to get nurses between government services, including the military service and the veterans hospital service, which we as private physicians in private hospitals have great difficulty in meeting, because these girls can begin their work in the initial grade in a veterans' hospital at perhaps $3600 a year with the prospect of rising to a level of $5500 a year in the highest position they can attain. That is serious competition for hospital administrators to meet in order to obtain a satisfactory number of nurses to staff our civilian hospitals. It is only by increasing the number who will be available that I can see any solution to the problem.

Perhaps one of the most serious problems with which we as a profession are confronted has to do with the care of the veteran. I am not too sure but that our present policy relative to the care of the veteran is a backdoor entry to socialized medicine. Only recently the Veterans Administrator released figures which were rather startling to me. At the end of 1950 there were 18,828,000 living veterans of our wars. In addition to that, there were 369,000 war widows and 288,000 dependent children, making a total, as you see, of nearly 20,000,000 who had the possibility of veterans' medical care. This number, unfortunately, as we all well know, is constantly increasing. We have no means of answering this at present, unless present policy is changed. Our legislators elected by us and other citizens have thought it necessary for one reason or another (it is difficult for me to see why) to provide this service for all of our veterans,

irrespective of whether the illness is service-connected and, in actual practice, irrespective of financial need.

Today we as a nation are not concerned with whether the disability of a veteran is related to his military service. At no time in the history of the world, at no time in the history of any nation, has the government become a Santa Claus to this extent. I do not believe that it is possible for any government to continue this, as well as other matters of equally expensive sort, and maintain its solvency. During the war, as you know, the various military services attempted to take care of all those in military service as well as their immediate dependents. Obviously, it is impossible for a government, certainly in the light of present facilities or in the light of present technical aid or physician aid, to contract to offer this extended medical service without some compulsory solicitation or some compulsory method of recruiting doctors to take care of this service.

At the present time we must congratulate the veterans medical service on the type of medicine which is being offered to those entering their hospitals. This is due to action of civilian physicians, such as Dr. Magnuson and others, and various deans' committees of our medical schools, who have advised and helped to staff these hospitals and certainly to advise them relative to medical care. For a time this was accepted by the government and those in charge of veterans' care, and veterans' hospitals were planned in metropolitan areas where they would be close to medical teaching, but this has not been continued. Those of us familiar with veterans' care in earlier years, before this influence of the proper type of civilian medical service in more recent years, know that the type of medical service which was offered under those circumstances was not of a high level. I would like to warn that unless this can be maintained under auspices not unlike those established in these recent years, the type of medical care that can be offered by the Veterans Administration services will drop, perhaps not to as low a level, but certainly it cannot be maintained at the present level.

When funds are available for veterans' care, as at present under these circumstances, obviously the various personnel of physicians, technicians, nurses and the like can be maintained, but when that level of service begins to drop or there is interference with the physician in this capacity, I assure you that those most competent to function in this capacity will no longer stay with it.

I would like to mention briefly a duty that many of us as physicians shirk, usually because we think we are too busy. The doctor is a citizen as well as a practitioner of medicine, and I am sure that even though we do cast our ballot and take individual responsibility as citizens, we do not take a proper place in politics and in political activity in our country. I think we should do so. I feel that I have done poorly in this regard, as I am sure many of you feel that you have, but when one thinks what can take place through the influence of doctors and their wives as evidenced in recent campaigns in Florida and in Minnesota and by the American Medical Association and organized medicine relative to defeating socialized medicine at least for the present time, one realizes that we have a real duty to perform if we are to accomplish all that we can to improve medical care.

There are doctors among us, one of whom addressed this Association in Chicago a few years ago, who have more than fulfilled their obligations as citizens. I refer to Dr. Walter H. Judd, a Representative from Minnesota. Dr. Judd, as a physician, has been able to influence legislation to a considerable extent and in that way I am sure has affected the practice of medicine, as have other physicians in public life in this way. This gives me an opportunity to mention another physician who has appeared on your program who has more than discharged his obligation as a citizen, Dr. John W. Cline, the present president of the American Medical Association.

I would like to call your attention, and particularly the attention of doctors' wives in the audience, to a new bill which has been introduced into our Congress by Senator Ives from New York. The Ives bill is a protection for our wives and for our children. This, to state it briefly, will offer us as physicians and lawyers and other professional men some relief with regard to present taxation, because of depletion of our ability to work. As you know, the oil industry permits 26 per cent for depletion of natural resources and I believe about 11 per cent for promotion. I do not think that the government or even Senator Ives has any thought of allowing us anything for promotion, but I think we need something for depletion, because over a relatively short period of a very active life, particularly in the light of present taxation which affects, of course, all of our citizens as well, we as physicians, with larger families (at least many of us) than the average of our country, do require some relief relative to the tax load. The major thing

that could be anticipated if the Ives bill were passed (and I think it will at least receive sympathetic consideration in the next Congress) would be to allow some funds to be put aside in pension schemes, in insurance, or some other form of security beyond the ordinary Social Security.

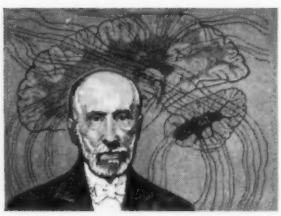
I urge you as physicians and as citizens to use your influence with your own Representatives in the Congress to call their attention to the justification for such a law.

One of the important considerations in this country today in medical practice has to do with the great increase in specialization that has taken place. Perhaps I have a right to speak about this as a specialist, but I do it with a sympathetic point of view relative to that important large body of physicians in this country, the general practitioners. This age of specialization which is so prominent in this country today has been an important one because it certainly has resulted in an improvement in medical care, but it has not in any way alleviated the problem of care of most of the patients who submit themselves to physicians. I think one of the things that has been responsible for this has been our medical schools. In their zeal for teaching science and for giving the new treatments and new disciplines and the specialized part of the various fields in medicine, I think they have held out research, they have held out teaching sometimes to the detriment of the training of the regular physician who will do the major portion of practice.

I think some of our medical schools today consider it their primary function to train the general practitioner to increase this large body that is so badly needed in this country. Unless we have an increased number of men who wish to follow that line of practice we will be unable to get the distribution of physicians that is so badly needed.

I have told you that I did not have any intent this evening to give a postgraduate course. I wanted solely to call your attention to certain things that were in my mind and I am sure in the minds of many of you relative to certain problems in American medicine today and what we can do about them. There must be a gradual change which will answer these problems. There will be a sufficient number of physicians, but I am sure that unless something is done we will leave a poor heritage for those who are coming after us. It must be our purpose to see that scientific medicine continues to be developed. The world looks to the United States to be sure that this is continued. We must continue our educational standards; we must continue our research, because it is by this means that we learn the new disciplines to improve medical practice; we must provide improved medical care at a reasonable cost, and we must provide the best distribution of physicians throughout this country that is possible without compulsion. We must guide the thinking of the public, and we must also guide the thinking of our elected representatives in our government. If we do so we will, perhaps, be better participating citizens in this country than we ever have been in the past.

CENTENARY OF RAMON Y CAJAL



JUST ONE CENTURY AGO, May 1, 1852, Santiago Ramon y Cajal was born in Petilla de Aragón, a small Spanish village. Son of a rural physician, his medical life started in 1873 with graduation from the Medical School of Zaragoza. After a short stay in Cuba as physician in the Spanish Army, he devoted his time to preparing for the competition for a chair in anatomy. These studies awakened in him his definitive vocation for histology. He was professor of anatomy in Valencia from 1883 to 1887, the date of his appointment to the chair of histology in the Medical School of Barcelona, where he started his research work, continued in Madrid from 1892.

His leading results, contained in the work of 1888 and 1889, were the real structure of nerve cells, the contact transmission of nervous stimuli and several features about the structure of the retina. His extraordinary scientific work won for him important rewards, such as honorary membership in most scientific academies and the Gold Medal of Helmholtz (not since awarded), Cajal being the sole biologist-physician having this medal. The acme came in 1906 with the grant of the Nobel Prize, shared with Professor Camilo Golgi. Reaching his seventieth birthday in 1922, Cajal was retired from his chair as well as from the position of Director of the National Institute of Health; he had been the first Director of this center. The Spanish government granted an important appropriation for a new Institute of Biology, named Cajal Institute by decision of the King. Cajal died in 1934.

The cover drawing was prepared for *Postgraduate Medicine* by Edmund Kopietz.

—*From* POSTGRADUATE MEDICINE'S *correspondent in Madrid.*